THE INTERNATIONAL MEETING
ON RESPIRATORY CARE INDONESIA

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HOMEPAGE
http://www.respina.org

STEERING COMMITTEE
Head Dept. of Pulmonology Fac. of Medicine Univ. of Indonesia
APSR - Representative for Indonesia
Governor of ACCP - Chapter Indonesia
Chairman of PERBRONKI
(Indonesian Society of Bronchoscopy)
Chairman of PDPI
(Indonesian Association of Pulmonologist)

Jakarta, July 24th 2019

No : 460/ Respina 21-Sec. / VII / 19

To :
Dr. dr. Mulyadi, Sp. P (K)

Dear **Dr. dr. Mulyadi, Sp. P (K),**

On behalf of the organizing committee, we would like to express our sincere thank you for your active participation as a Speaker in **The 21st International Meeting on Respiratory Care Indonesia (Respina) 2019**, which is held on 25th – 27th July 2019 at JW Marriott Hotel, Jakarta. We are really grateful for your attention and support to make our conference fruitful this year.

We also would like to apologize for any inconvenience during the preparation symposium. We have tried to do our best for our colleagues. Please do not hesitate to contact us if there are any suggestions, critics or information.

Thank you for your kind attention and cooperation. It is our sincere wish to see you again in the near future.

Sincerely,

Pompini Agustina, MD
Chairperson of the Organizing Committee of Respina 2019

Cc:
- President of The Society of Respiratory Care Indonesia (RESPINA)
- Archives

THE 21ST INTERNATIONAL MEETING ON RESPIRATORY CARE INDONESIA (Respina) 2019

RS Penyakit Infeksi Prof. Dr. Sulianti Saroso, Jakarta, 24th - 25th July 2019

JW Marriott Hotel, Jakarta, 25th - 27th July 2019

"Facing the Uncertainty" Focus on: Respiratory Care in Disaster

Certificate of Attendance

MULYADI

AS

SPEAKER

SK PB IDI No. : 0453/PB/A.4/06/2019

PARTICIPANT : 12 SKP, PEMBICARA : 14 SKP, MODERATOR : 4 SKP, PANITIA : 3 SKP

Pompini Agustina, MD

Chairperson of Respina 2019

"Facing the Uncertainty"

Focus on: Respiratory Care in Disaster

The 21st International Meeting on Respiratory Care Indonesia (Respina) 2019

WORKSHOPS

RSPI Prof. Dr. Sulianti Saroso, Jakarta
24th – 25th July 2019

JW Marriott Hotel, Jakarta
25th July 2019

SYMPOSIUM

JW Marriott Hotel, Jakarta
26th – 27th July 2019

Proceeding E-book

Editor
Pompini Agustina, MD
Wahyuningsih, MD
Anitta Florence Stans Paulus, MD
Asri Liqditta Bies, MD
Devina Arrandhikasari, MD

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Jakarta, Indonesia
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Jl. Matraman No. 30 E Central Jakarta-INDONESIA

http://www.respina.org

First published 2019
Printed in the Jakarta at the Society of Respiratory Care Indonesia (RESPINA)

ISBN

Contents

Welcome Message

Dear Colleagues,

On behalf of 21st Respina 2019 Organizing Committee, we extend a warm welcome to all participants to attend The International Meeting of Respiratory Care Indonesia which will be held on 24th – 27th July 2019 in JW Marriot Hotel, Jakarta. Respina has been organized since 1998 as one of the largest Respiratory Care event in Southeast Asia.

In its development, Indonesia has experienced many natural disasters such as: the eruption of Mount Merapi, forest fires, tsunamis and earthquakes which have an impact on respiration problems which are serious enough so that appropriate and true handling is needed from various disciplines.

Featuring the theme **"Facing the Uncertainty" Focus on: Respiratory Care in Disaster".**, Respina 2019 tries to solve this problem by inviting experts in their respective fields

There will be seven workshop's, in which we collaborate with National Agency of Food and Drugs Control Badan Pengawas Obat dan Makanan/BPOM) and National Institute of Health, Research, and Development (Badan Penelitian dan Pengembangan Kesehatan/Balitbangkes).

The scientific symposia will gather a number of prominent experts presenting plenary lectures as well as many more specific sessions. We do hope to create the attractive and knowledgeable conference. RespiQuizz will provide the chance to see the capabilities and competitiveness between students from various medical faculties in Indonesia. Last but not least, the novel studies and advancements in respiratory care will be presented by various specialists in poster session.

In conclusion, The 21st Respina will offer great opportunities for the participants to discuss recent topics regarding respiratory care in their respective expertise's and share their experiences in daily practice.

We are looking forward to see you in Jakarta to enjoy the excitement of this scientific meeting

Warm Regards,

Pompini Agustina, M.D.
Chairperson of the Organizing Committee

About Respina 2019

Respiratory Care Indonesia (Respina) is an annual international meeting in Indonesia on respiratory care. Respina is a result of collaboration of five pillars, which are Department of Pulmonology and Respiratory Medicine Faculty of Medicine University of Indonesia, American College of Chest Physician-Indonesia Chapter, Asian Pacific Society of Respirology, Indonesia Society of Bronchoscopy and Indonesian Society of Respirology, in answering the global problem of respiratory care. The mission of the meeting is to bring the up-to-date and latest information of respiratory care and as media of collaboration to each respiratory care practitioners in cooperative spirit.

Starting on 2006, Respina is proudly joined by societies that shared the same interest particularly in respiratory care, and they are as follows:

- Indonesian Society of Respirology
- Indonesian Association of Thoracic and Cardiovascular Surgeons
- Indonesian Radiological Society
- Indonesian Neurological Association
- Indonesian Heart Association
- The Indonesian Society of Anesthesiology and Intensive Therapy
- The Indonesian of Physical Medicine and Rehabilitation Association
- Indonesian Pediatric Society
- The Indonesian Otorhinolaryngological Head and Neck Surgery Society

Four other professional organizations joined Respina in 2011, they are:

- Indonesian Association of Clinical Pathologists
- The Indonesian Physician of community medicine and Public Health Association
- Indonesian Sports Medicine Association
- Indonesian Society for Clinical Microbiology

Respina 2019 is the 21st meeting we have been conducting and during the years, Respina has become one of the major respiratory events in Indonesia and gained greater and still growing interest from physicians across the regions, particularly from our colleagues in Southeast Asia.

Society by:

Organizing Committee

PILLAR
- Department of Pulmonology and Respiratory Medicine Faculty of Medicine University of Indonesia
- American College of Chest Physician (ACCP) - Indonesian Chapter
- Asian Pacific Society of Respirology (APSR) Representative for Indonesia
- The Indonesia Society of Bronchoscopy (PERBRONKI) Indonesia
- Indonesian Society of Respirology (PDPI)

HONORARY CHAIR
- Prof. Hadiarto Mangunnegoro, MD, FCCP
- Venugopal S. Reddy, MD, FCCP

BOARD COMMISION
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Secretary	:	Santi Rahayu Dewayanti, MD
Treasurer	:	Linda Masniari, MD
		Feni Fitriani Taufik, MD

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RESPIQUIZZ
- Kasum Supriadinata, MD
- Shaogi Syam, MD
- Jaka Pradipta, MD
- Widhy Yudistira, MD
- Dian Prastiti, MD

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- Vinci Edy Wibowo, MD
- Raden Fajar Prosojo Utomo, MD
- Haruyuki Dewi Faisal, MD

FINANCE & FACILITIES
- Retno Wihastuti, MD
- Amira Anwar, MD
- Bambang Heru, MD

INVITED SPEAKERS

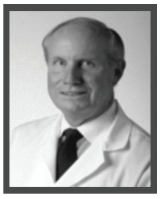
- Amanda Piper (AUS)
- Chiaki Toida (JPN)
- Gary Lee (AUS)
- Jennifer Ann Mendoza-Wi (PHI)
- Lim Chong Hee (SIN)
- Martin J. Tobin (USA)
- Menaldi Rasmin (INA)
- Naoto Morimura (JPN)
- Philip Eng (SIN)
- Rodolfo R. T. Bigornia (PHI)
- T. Agasthian (SIN)

Faculty Members

Cesare Gregoretti,
Director of General Intensive Care Unit and Anesthesiology Service Orthopedic and Trauma Center of Turin, Italy.
Independent referee of international journals such as Intensive Care Medicine, European Respiratory Journal, Respiratory Medicine.
Professor of Intensive Care and Anesthesiology at the postgraduate school, University of Turin and Novara Italy.
Speaker and chairman at main national and international conventions in the field of mechanical ventilation and an Invited Professor at Tuft University in Boston and Temple University in Philadelphia.

Jennifer Ann Mendoza-Wi
Full Professor Lyceum Northwestern FQ Duque College of Medicine, Dagupan City.
International Governor, American College of Chest Physicians, Philippine Chapter.

Martin Tobin
Professor of Medicine, Pulmonary and Critical Care Medicine.
Division Director, Pulmonary & Critical Care Medicine Special Interests: Acute Respiratory Failure.
Neuromuscular Control of Breathing
Mechanical Ventilation.



Neil Ross Macintyre, JR
Professor of Medicine
Duke University Medical Center
Chief of Clinical Services
Division of Pulmonary and Critical Care Medicine
Medical Director of Respiratory Care Services,
Pulmonary Function Laboratory, and
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Duke University Medical Center
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Nicolino Ambrosino
Appointed Professor of Universities of Pisa, Pavia, Firenze, Trieste, Milano
Professor Head, Pulmonary Department, Cardio-Thoracic Department, University Hospital, Pisa.
Head, Pulmonary Unit, Respiratory Intensive Care. Cardio-Thoracic Department, University Hospital, Pisa.
Head, Pulmonary Rehabilitation and Weaning Center, Auxilium Vitae, Volterra
Scientific Director of Auxilium Vitae, Volterra
Former Head : Pulmonary Division and Intermediate Intensive Care. Medical Center of Gussago, S. Maugeri Foundation

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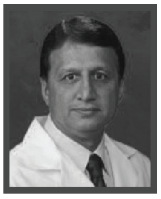
Ospedale Maggiore della Carità in Novara (Italy).
Head of the Intensive Care Unit of the Department of Anesthesia and Intensive Care.
Teaches at the School of Anesthesiology and Intensive Care of The University of the Oriental Piedmont.

Richard Wayne Light
Practices Internal Medicine, Pulmonary Disease near Nashville, TN.
Professor of Medicine, Vanderbilt University, Nashville.
Professor Emeritus of Medicine, University of California at Irvine



Venugopal S Reddy
Associate Professor of Anesthesia and Critical Care Medicine.
Divisional Director Critical Care Medicine
Director of Surgical Anesthesia Intensive Care Unit.
Director of Mortality and Morbidity meeting Penn State College of Medicine and Hershey Medical Center, Hershey, USA.

Scientific Program

DAY 1 FRIDAY, 26th July 2019

REGISTRATION			
MORNING SYMPOSIUM 1 Lung Problem in Enviromental High Risk Disaster : Children	**MORNING SYMPOSIUM 2** Lung Problem in Enviromental High Risk Disaster : Adult	**MORNING SYMPOSIUM 3** Lung Problem in Enviromental High Risk Disaster : Community	
PLENARY SESSION 1 Webinar : The Series of Natural Disaster			
OPENING CEREMONY			
SATELLITE SYMPOSIUM 1 Asthma & COPD in High Risk Disaster Area	**SATELLITE SYMPOSIUM 2** Managing Recurrent RTI in Evacuated Victims With Immunotherapy	**SATELLITE SYMPOSIUM 3** Medical Role in Disaster Management	
LUNCH & FRIDAY PRAYING			
MASTER CLASS 1 RESPIRATORY CARE : In the Event of Volcano Eruption & Fire	**MASTER CLASS 2** RESPIRATORY CARE : In the Event of Tsunami & Floods	**MASTER CLASS 3** RESPIRATORY CARE : In the Event of Earthquake & Landslide	
MEET THE EXPERT 1 RESPIRATORY FAILURE : Volcano Eruption and Fire	**MEET THE EXPERT 2** Impact Acute and Chronic to Respiratory Post Disaster	**MEET THE EXPERT 3** Emergency Invasive Care in Disaster Situation	
SATELLITE SYMPOSIUM 4 Acute Impact on Natural Disaster	**SATELLITE SYMPOSIUM 5** Planning and Preparedness in Disaster Management	**SATELLITE SYMPOSIUM 6** Physical Health Care in Disaster Area	**FREE PAPER & POSTER SESSION**
STUDIUM GENERALE Art of Medicine in Millennial Era: Continued Innovation is the Best Way to Beat the Competition			

Scientific Program

DAY 2 SATURDAY, 27st July 2019

REGISTRATION		
MORNING SYMPOSIUM 4 Supporting Exam of Respiratory Disease on Disaster	**MORNING SYMPOSIUM 5** Respiratory Problem in Tsunami Victims	**MORNING SYMPOSIUM 6** Critical Care in Disaster
PLENARY SESSION 2 From Upstream to Downstream : Disaster		
RespiQuizz		
LESSON'S LEARNED Tsunami Lung		
SATELLITE SYMPOSIUM 7 Preparing for Natural Disaster in Airway Disease	**SATELLITE SYMPOSIUM 8** COPD in Disaster Situation	**SATELLITE SYMPOSIUM 9** Respiratory Infection in Disaster
LUNCH & PRAYING		
LUNCH SYMPOSIUM 1 Tumor Event Following Disaster	**LUNCH SYMPOSIUM 2** The Strategy on Pulmonary Infection Prevention, Detection & Treatment in Disaster	
SATELLITE SYMPOSIUM 10 Early Response During & After Disaster	**SATELLITE SYMPOSIUM 11** Challenges Facing Lung Problems Post Disaster	**SATELLITE SYMPOSIUM 12** Mucous Clearence Management
SUMMARY "Facing the Uncertainty" Focus on: Respiratory Care in Disaster		
CLOSING CEREMONY		

Scientific Schedule

DAY 1 FRIDAY, 26TH JULY 2019

REGISTRATION
07.00 - 07.30

MORNING SYMPOSIUM 1
Lung Problem in Enviromental High Risk Disaster : Children

07.30 - 07.50	Acute Respiratory Problem in Children *Wahyuni Indawati (INA)*
07.50 - 08.10	Chronic Respiratory Problem in Children *Bambang Supriyatno (INA)*
08.10 - 08.15	Discussion

MORNING SYMPOSIUM 2
Lung Problem in Enviromental High Risk Disaster : Adult

07.30 - 07.50	Respiratory Problem in Adult: Upper Airway *Syahrial M. Hutauruk (INA)*
07.50 - 08.10	Biomolecular Aspect of Chronic Obstructive Pulmonary Disease *Johny Anwar (INA)*
08.10 - 08.15	Discussion

MORNING SYMPOSIUM 3
Lung Problem in Enviromental High Risk Disaster : Community

07.30 - 07.50	Epidemiology of Diseases in Volcanic Area *Trevino Pakasi (INA)*
07.50 - 08.10	Lung Problem in Smoke Fire Event *Feni Fitriani Taufik (INA)*
08.10 - 08.15	Discussion

PLENARY SESSION 1
Webinar : The Series of Natural Disaster

08.15 - 08.40	Health Policy in National Disaster Management *Ahmad Yurianto (Crisis Center KEMENKES - INA)*
08.40 - 09.10	Clinical Science: The Consensus - Controversy Conundrum *Martin J. Tobin (USA)*
09.10 - 09.35	Medical Team & Patient Safety in Disaster Area *Daeng M. Faqih (PB IDI - INA)*

OPENING CEREMONY
09.35 - 10.30

SATELLITE SYMPOSIUM 1
Asthma & COPD in High Risk Disaster Area

10.30 - 10.55	COPD *Linda Masniari (INA)*
10.55 - 11.20	Asthma *Hadiarto Mangunnegoro (INA)*
11.20 - 11.30	Discussion & Quizz

SATELLITE SYMPOSIUM 2
Managing Recurrent RTI in Evacuated Victims With Immunotherapy

10.30 - 10.55	Pharmacological Point of View of Immunotherapy for Recurrent RTI in Evacuated Victims *Purwantyastuti Ascobat (INA)*
10.55 - 11.20	Bacterial Lysate : In Prevention of Lung Disease *Menaldi Rasmin (INA)*
11.20 - 11.30	Discussion

SATELLITE SYMPOSIUM 3
Medical Role in Disaster Management

10.30 - 10.55	Focus on Disaster Medicine *Naoto Morimura (JPN)*
10.55 - 11.20	Management of COPD in Disaster *Faisal Yunus (INA)*
11.20 - 11.30	Discussion

LUNCH & FRIDAY PRAYER
11.30 - 13.00

MASTER CLASS 1
RESPIRATORY CARE : In the Event of Volcano Eruption & Fire

13.00 - 13.25	Respiratory Care on Volcano Eruption and Fire *Ngakan Putu Parsama Putra (INA)*
13.25 - 13.50	Respiratory Care on Volcano Eruption and Fire *Rodolfo R.T. Bigornia (PHI)*
13.50 - 14.00	Discussion

MASTER CLASS 2
RESPIRATORY CARE : In the Event of Tsunami & Floods

13.00 - 13.25	Respiratory Care on Tsunami & Flood *Chiaki Toida (JPN)*
13.25 - 13.50	Respiratory Care Tsunami & Flood *Mulyadi (INA)*
13.50 - 14.00	Discussion *Chiaki Toida (JPN),Naoto Morimura (JPN), Mulyadi (INA)*

Scientific Schedule

MASTER CLASS 3
RESPIRATORY CARE : In the Event of Earthquake & Landslide

13.00 - 13.25 Respiratory Care on Earthquake & Landslide
Jennifer Ann Mendoza-Wi (PHI)
13.25 - 13.55 The Physiologic Basis of Mechanical Ventilation
Martin J. Tobin (USA)
13.55 - 14.00 Discussion

MEET THE EXPERT 1
RESPIRATORY FAILURE : Volcano Eruption and Fire

14.00 - 14.25 Air Polution in Disaster Area
Mukhtar Ikhsan (INA)
14.25 - 14.50 Inhalation Injury
Feni Fitriani Taufik (INA)
14.50 - 15.00 Discussion

MEET THE EXPERT 2
Impact Acute and Chronic to Respiratory Post Disaster

14.00 - 14.15 Community Acquired Pneumonia : From Moderate to Severe
Santi Rahayu Dewayanti (INA)
14.15 - 14.40 Management of Invasive Fungal Infections: Focus on Respiratory Fungal Infections
Anwar Jusuf (INA)
14.40 - 14.55 ILD Early Detection
Fanny Fachrucha (INA)
14.55 - 15.00 Discussion

MEET THE EXPERT 3
Emergency Invasive Care in Disaster Situation

14.00 - 14.25 Penetrating Cardiac Injury in Surabaya
Paul Tahalele (INA)
14.25 - 14.50 Latest Treatments for Chronic Pulmonary Thromboembolic Hypertension
Lim Chong Hee (SIN)
14.50 - 15.00 Discussion

SATELLITE SYMPOSIUM 4
Acute Impact on Natural Disaster

15.00 - 15.20 Impact Disaster Neurology System
Rakhmad Hidayat (INA)
15.20 - 15.40 Analgesia Considerations for Patients with Respiratory Problems
I Putu Pramana Suarjaya (INA)
15.40 - 16.00 Pneumothorax
Gary Lee (SIN)
16.00 - 16.05 Discussion

SATELLITE SYMPOSIUM 5
Planning and Preparedness in Disaster Management

15.00 - 15.25 Management of Acute Respiratory Emergency Cases
Tri Agus Yuarsa (INA)
15.25 - 15.50 Management of Respiratory Problems After Disasters
Arif Santoso (INA)
15.50 - 16.05 Discussion

SATELLITE SYMPOSIUM 6
Physical Health Care in Disaster Area

15.00 - 15.20 Focused on Healthy Subjects
Zaini (INA)
15.20 - 15.40 Pulmonary Rehabilitation of Respiratory Disease on Disaster
Siti Chandra Widjanantie (INA)
15.40 - 16.00 Prevention of Chronic Lung Disease in Disaster Area
Pompini Agustina (INA)
16.00 - 16.05 Discussion

FREE PAPER
15.00 - 16.05

POSTER SESSION
15.00 - 16.05

STUDIUM GENERALE
16.05 - 17.00 Art of Medicine in Millennial Era: Continued Innovation is the Best Way to Beat the Competition
Jennifer Ann Mendoza-Wi (PHI)

Scientific Schedule

DAY 2 SATURDAY, 27th JULY 2019

REGISTRATION
07.00 - 07.30

MORNING SYMPOSIUM 4
Supporting Exam of Respiratory Disease on Disaster

07.30 - 07.50	Radiologic Findings in Neonatal Respiratory Distres *Wuri Suryandari (INA)*
07.50 - 08.10	Laboratory Parameter & Method of Choice for Health Problem After Natural Disease *Ida Parwati (INA)*
08.10 - 08.30	Embolisation in Hemoptisis *Andi Darwis (INA)*
08.30 - 08.35	Discussion

MORNING SYMPOSIUM 5
Respiratory Problem in Tsunami Victims

07.30 - 07.50	Interventional Procedure in Adults *Rita Rogayah (INA))*
07.50 - 08.10	In Children *Tjatur K. Sagoro (INA)*
08.10 - 08.30	Pleural Infection: What is New? *Gary Lee (AUS)*
08.30 - 08.35	Discussion

MORNING SYMPOSIUM 6
Critical Care in Disaster

07.30 - 07.50	Pain Management in Critically Ill *Faisal Muchtar (INA)*
07.50 - 08.10	Critical Care in Disaster : Thoracic Trauma *Susan H. Meity (INA)*
08.10 - 08.30	Cardiac Resusitation *Berlian Idriansyah Idris (INA)*
08.30 - 08.35	Discussion

PLENARY SESSION 2
From Upstream to Downstream : Disaster

08.35 - 08.55	Do We Need a Disaster Curriculum in Medical Education? *Nurdin Perdana (Asosiasi Institusi Pendidikan Kedokteran Indonesia - INA)*
08.55 - 09.15	National Disaster Management Policy *Bagus Tjahyono (BNPB - INA)*
09.15 - 09.35	The Strength of Multidicipline in Disaster Emergency Response *Wahyuningsih Suharno (INA)*

RESPIQUIZZ
09.35 - 10.35

LESSON'S LEARNED
Tsunami Lung
10.35 - 11.35

SATELLITE SYMPOSIUM 7
Preparing for Natural Disaster in Airway Disease

11.35 - 11.55	Asthma *Jennifer Ann Mendoza-Wi (PHI)*
11.55 - 12.15	Time to Shift to New Paradigm in Asthma Management *Pradjnaparamita (INA)*
12.15 - 12.35	COPD Management Care Post Disaster *Budhi Antariksa (INA)*
12.35 - 12.40	Discussion

SATELLITE SYMPOSIUM 8
COPD in Disaster Situation
Moderator : Wahyuningsih Suharno (INA)

11.35 - 11.55	Mastering the Art of COPD: How to Diagnose in Disaster Situation *Retno Wihastuti (INA)*
11.55 - 12.15	Increasing LABA/LAMA Role in COPD Management: Working on Disaster Situation, Adaptation from GOLD 2019 *Faisal Yunus (INA)*
12.15 - 12.35	Prevention of COPD Exacerbation *Amanda Piper (INA)*
12.35 - 12.40	Discussion

SATELLITE SYMPOSIUM 9
Respiratory Infection in Disaster

11.35 - 11.55	Rehabilitation Post Intervention *Anita Ratnawati (INA)*
11.55 - 12.15	Microbiology Pattern in New Era of Antimicrobia Resistance *Kuntaman (INA)*
12.15 - 12.35	Respiratory Failure due to Infection in Disaster *Erlina Burhan (INA)*
12.35 - 12.40	Discussion

LUNCH & PRAY
12.40 - 13.30

Scientific Schedule

LUNCH SYMPOSIUM 1
Tumor Event Following Disaster

13.30 - 13.50 The Likelihood of Tumors Occurrence
Marlinda Adham (INA)
13.50 - 14.10 Mesothelioma & Asbestosis
T. Agasthian (SIN)
14.10 - 14.15 COUGHING ETHICS EXERCISES
14.15 - 14.35 Lung Cancer : The Long Term Effect After Lung Injury in Disaster
Jamal Zaini (INA)

LUNCH SYMPOSIUM 2
The Strategy on Pulmonary Infection Prevention, Detection & Treatment in Disaster

13.30 - 13.50 Early Management for Patients in Volcano Area
Jennifer Ann Mendoza-Wi (PHI)
13.50 - 14.10 ICU Setting in Disaster Area
Rodolfo R. T. Bigornia (PHI)
14.10 - 14.15 COUGHING ETHICS EXERCISES
14.15 - 14.35 Continuing Therapy Post Disaster
Rita Rogayah (INA)

SATELLITE SYMPOSIUM 10
Early Response During and After Disaster

14.35 - 15.00 Victims With and Without Disability
Adhityawarman (INA)
15.00 - 15.25 Emergency Medical Respiratory Team
Dewi Puspitorini (INA)
15.25 - 15.35 Discussion

SATELLITE SYMPOSIUM 11
Challenges Facing Lung Problems Post Disaster

14.35 - 15.00 Pulmonary Infection after Disaster : How to Choose Antibiotics
Erlina Burhan (INA)
15.00 - 15.25 ECMO for Viral Pneumonia
Philip Eng (SIN)
15.25 - 15.35 Discussion

SATELLITE SYMPOSIUM 12
Mucous Clearence Management

14.35 - 15.00 Interventional Procedure in Mucous Clearance Following Natural Disaster
Prasenohadi (INA)
15.00 - 15.25 Classic to Advance Airway Clearance Technique
Nury N. Handikin (INA)
15.25 - 15.35 Discussion

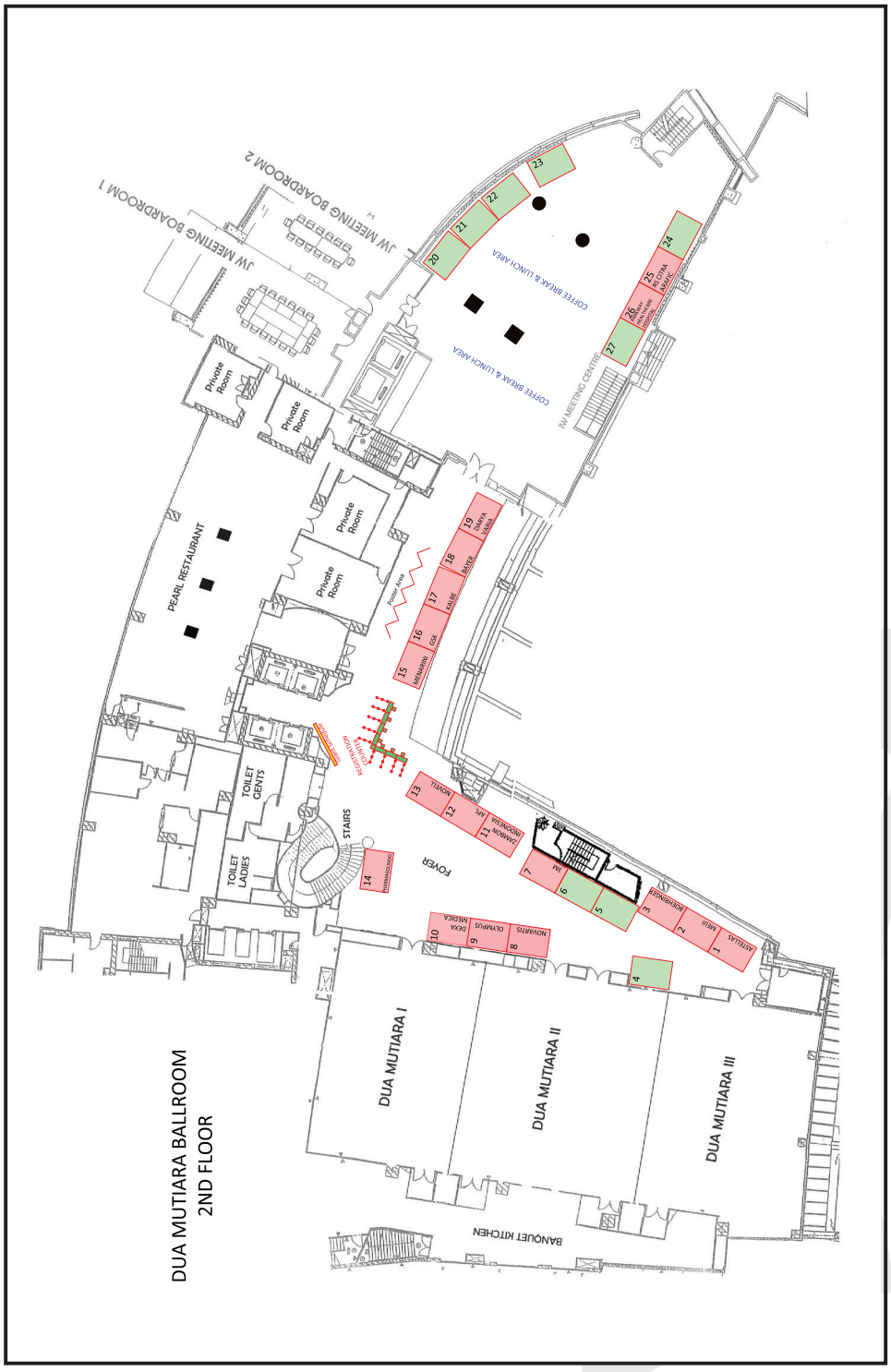
SUMMARY

16.35 - 16.05 "Facing the Uncertainty" Focus on: Respiratory Care in Disaster
Menaldi Rasmin (INA)

CLOSING CEREMONY

16.05 - 16.25

Exhibition Floor Plan



Exhibitor List

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1.	Astellas Pharma Indonesia
2.	Meiji Indonesia
3.	Boehringer Ingelheim
4.	3M
5.	Novartis Indonesia
6.	Olympus
7.	Dexa Medica
8.	Zambon Indonesia
9.	Anugerah Pharmindo Lestari
10.	Novell Par Pharmaceuticals
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12.	Glaxo Smith Kline
13.	Menarini
14.	Kalbe Farma
15.	Bayer Indonesia
16.	Darya Varia
17.	RS Citra Arafiq Medika

Acknowledgement

1. Novartis Indonesia
2. Glaxo Smith Kline
3. Astra Zeneca Indonesia
4. Boehringer Ingelheim
5. Actavis Indonesia
6. Pfizer Indonesia
7. Zambon Indonesia
8. Dexa Medica
9. Novell Par Pharmaceuticals
10. Pharmasolindo
11. Menarini
12. Anugerah Pharmindo Lestari
13. Darya Varia
14. Astellas Pharma Indonesia
15. Meiji Indonesia
16. Kalbe Farma
17. Bayer Indonesia
18. Prodia Laboratories
19. Parkway Hospitals Singapore
20. RS Citra Arafiq Medika
21. Hutama Karya

Full Paper Contents

Full Paper Contents

Full Paper Contents

FRIDAY, 26th JULY 2019

BIOMOLECULAR ASPECT OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Johny Anwar
RSUP Dr. Mohammad Hoesin Palembang

ABSTRACT

1. Pendahuluan

Paru merupakan organ yang paling sering terpapar dengan oksidan atau radikal bebas yang dapat menyebabkan kerusakan pada protein, lipid, dan asam nukleat. Kebiasaan merokok merupakan faktor utama yang sangat penting sebagai penyebab terjadinya emfisema. Walaupun tidak semua perokok menderita emfisema, tetapi 15 - 20% perokok menderita penyakit ini (Honig dkk, 2001; Barnes dkk, 2003; Barnes, 2004).

Patogenesis terjadinya emfisema akibat paparan asap rokok yang kronik tidak sepenuhnya diketahui, diduga akibat penarikan sel inflamasi ke saluran napas perifer, pelepasan enzim elastolitik dan tidak efektifnya proses perbaikan kembali *(repair)* serat elastin dan komponen matriks yang lain (Shapiro, 1999).

2. Organ Paru

Paru merupakan organ pernapasan yang berfungsi untuk mensuplai oksigen ke seluruh organ tubuh. Sistem pernapasan mempunyai permukaan yang luas tetapi sangat rentan terhadap perubahan dan variasi lingkungan. Paru merupakan organ yang sering terpapar oleh polutan yang terdapat di udara dan berisiko tinggi terhadap kerusakan oksidatif karena terpapar langsung dengan zat toksik yang berasal dari asap kendaraan bermotor, asap rokok dan infeksi. Jika jumlah oksidan yang masuk berlebihan dan pertahanan antioksidan yang tidak efektif dapat menyebabkan terjadinya kerusakan paru (Donno dkk, 2000). Berikut ini akan dibahas mengenai anatomi paru dan matrik ekstrasel.

2.1. Anatomi Paru

Saluran napas dibagi menjadi 2 bagian yaitu zona penghantar dan zona pernapasan. Zona penghantar berfungsi sebagai penghantar aliran udara sedangkan zona pernapasan berfungsi untuk pertukaran gas. Saluran napas dimulai dari trakea kemudian bercabang menjadi bronkus kanan dan kiri. Bronkus bercabang secara dikotomis sebanyak 23 kali. Percabangan bronkus yang tidak mengandung tulang rawan disebut bronkiolus. Cabang terakhir dari bronkiolus yang tidak mengandung alveolus disebut bronkiolus terminalis. Zona pernapasan dimulai dari bronkiolus respiratorius, duktus alveolaris, sakus alveolaris dan berakhir pada alveolus. Udara pada alveolus akan memasuki kapiler melalui membran yang sangat tipis yaitu membran alveolo-kapilaris. Paru mengandung sekitar 300 juta alveolus dengan luas permukaan sekitar 143 m2. Dinding alveolus mengandung beberapa macam sel yaitu sel pneumosit tipe I yang meliputi 95% permukaan alveolus, sel pneumosit type II yang berfungsi memproduksi surfaktan dan makrofag yang berfungsi untuk fagositik dan imunologi. Pneumosit tipe I juga mempunyai sifat fagositik tetapi lebih rentan jika dibandingkan pneumosit tipe II. Sel pneumosit berhubungan dengan makromolekul yang terdapat diluar sel dan membentuk matriks ekstrasel (Amin, 2005).

2.2. Matriks ekstrasel (MES)

Pada organisme multiseluler, sel saling berhubungan melalui makromolekul yang disebut matriks ekstrasel yang dibentuk dari protein dan polisakarida. Interaksi antara sel dan matriks ekstrasel mempunyai peranan yang penting pada fisiologi, siklus kinetik sel serta proses terjadinya reorganisasi dan remodeling matriks ekstrasel. Matriks ekstrasel terutama mengandung serat protein yang terdapat didalam gel polisakarida dan diperlukan untuk integritas paru. Makromolekul yang menyusun matriks ekstrasel terutama adalah

kolagen dan proteoglikan sedangkan makromolekul yang lainnya adalah elastin, fibronektin dan laminin. Sebagian besar kolagen pada paru adalah kolagen tipe I dan tipe II (Suki, 2005. kutipan Amin, 2005). Matriks ekstrasel mengalami penggantian yang terus menerus sebanyak lebih dari 10% perhari (Eichelberg dkk, 1999). Dalam keadaan normal terdapat keseimbangan antara sintesis dan degradasi matriks ektrasel untuk menjaga keseimbangan fisiologis. Keseimbangan ini dikontrol dengan baik oleh 3 mekanisme regulasi yaitu ;

1. Sintesis *denovo* dan degradasi komponen MES seperti kolagen.
2. Degradasi proteolitik MES oleh matriks metalloproteinase (MMP)
3. Pencegahan terhadap aktivasi MMP oleh antiprotease endogen seperti TIMPs

Kerusakan pada MES seperti serat elastin akibat asap rokok menyebabkan perubahan struktur dan terjadinya emfisema. (Betsuyaku dkk, 1999. Foronjy dkk, 2001).

2.2.1 Kolagen

Sampai saat ini telah diketahui lebih dari 20 tipe kolagen dan kebanyakan adalah kolagen tipe I, II, III, V dan XI. Kolagen tipe I merupakan yang terbanyak (90%). Masing masing tipe kolagen mempunyai sifat yang berbeda. Kolagen merupakan penahan beban yang sangat penting pada duktus dan dinding alveoli. Sekitar 25% dari protein yang ada didalam matriks ekstrasel adalah kolagen.

Struktur kolagen berbentuk heliks rangkap 3 *(triple heliks)* yang dibentuk oleh 3 rantai polipeptida. Ketiga rantai polipeptida ini membetuk superhelix yang memutar ke kanan dan membentuk struktur yang kaku mirip batang dengan panjang 300 nm dan diameter 1,5 nm. Kolagen tipe I, II dan III membentuk polimer yaitu fibril kolagen yang panjang, halus dan mirip kabel. Fibril kolagen ini membentuk serat kolagen. Sedangkan kolagen tipe IV dan V, terdapat pada lamina basalis tetapi tidak membentuk fibril. Kekurangan serat kolagen dapat menyebabkan jaringan menjadi lebih rapuh dan mudah robek (Suki dkk. 2005, Amin, 2005).

2.2.2 Elastin

Serat elastin terdiri dari elastin dan mikrofibril. Mikrofibril yang terutama adalah fibrilin dan fibulin. Elastisitas mikrofibril ini masih kontroversial dan peranannya pada elastisitas paru belum banyak dipelajari. Elastin merupakan suatu senyawa protein polimerik yang membentuk serat elastin dan berfungsi untuk mempertahankan elastisitas dari paru. Struktur 3 dimensi molekul serat elastin belum diketahui dengan baik.

Elastin terdiri dari polipeptida yang fleksibel, melekat pada kolagen dan mudah memanjang hingga lebih dari 200% (Suki dkk,2005). Elastin terdiri dari monomer tropoelastin yang saling berikatan melalui ikatan silang dan membentuk jaringan yang terdiri dari berbagai serat tersusun menjadi berbagai lembaran. Serat elastin dapat meregang dan mengkerutkan suatu jaringan. Serat elastin juga dapat memelihara patensi dan kelenturan dinding alveolar (Suradi, 2004).

Kerusakan elastin akibat elastase menyebabkan paru kehilangan elastisitasnya *(lost of elasticity)* dan kerusakan ini tidak bisa di regenerasi dalam bentuk semula (Barnes dkk, 2003). Instilasi elastase pankreas kedalam saluran napas menyebabkan terjadi kerusakan elastin paru tetapi kemudian terjadi proses resintesis elastin dalam 2 bulan tetapi elastin yang terbentuk tidak teratur dan tidak dapat mengembalikan integritas anatomi paru (Kimbel, 1980).

2.2.3 Proteoglikan

Proteoglikan merupakan makromolekul yang terdiri dari 2 komponen yaitu protein dan karbohidrat yang saling berikatan melalui ikatan kovalen. Pada proteoglikan terdapat komponen karbohidrat yang lebih dominan sedangkan pada glikoprotein yang lebih dominan adalah protein. Komponen karbohidrat pada glikoprotein adalah polisakarida yang berbentuk seperti lendir *(mucoid)* sehingga disebut mukopolisakarida atau nama lainnya yaitu glikosaminoglikan. Polisakarida ini membentuk rantai panjang yang tidak bercabang dan terdiri dari unit disakarida yang berulang salah satunya adalah gula amino (D-Glukosamin) atau L-galaktosamin, sedangkan disakarida yang lainya adalah galaktosa atau asam uronat.

Kandungan utama dari MES adalah glukosaminoglikan yang terdiri dari asam hialuronat, khondrointin sulfat, dermatan sulfat dan keratan sulfat.

Proteoglikan utama pada paru adalah heparan sulfat dan khondrointin sulfat (Suki dkk, 2005). Sifat proteoglikan ini tidak lentur, hidrofilik, mengisi sebagian besar volume MES dan membentuk gel yang berfungsi untuk melindungi jaringan terhadap kompresi. Proteoglikan juga dapat memudahkan migrasi sel pada waktu morfogenesis dan reparasi akibat jejas (Amin, 2005).

2.2.4 Fibronektin

Fibronektin merupakan glikoprotein yang *multi-domain* dengan berat molekul tinggi dan mengandung 5% karbohidrat. Fibronektin merupakan glikoprotein yang *multi-domain*, terdapat dalam bentuk yang larut dalam plasma dan yang tidak larut yaitu pada jaringan ikat, membran basal dan dapat mengikat berbagai komponen kolagen, fibrin, heparin dan aktin. Fibronektin mempunyai 3 tipe domain yaitu tipe I, II dan III. Fibronektin domain tipe II melekat pada kolagen.

Fibronektin mempunyai 2 rantai polipeptida yang melekat melalui ikatan disulfida dan melipat menjadi 5–6 unit fungsional. Unit fungsional ini berikatan dengan molekul MES yang lain seperti kolagen, fibrin, heparan sulfat dan membran sel melalui integrin. Fibronektin berfungsi untuk membantu ikatan antar sel, migrasi sel, mempertahankan bentuk dan stabilitas sel dengan MES, membentuk jejaring yang berfungsi sebagai plasma opsonin. Fibronektin juga berperan pada penyembuhan luka, adhesi sel, koagulasi darah, differensiasi dan migrasi sel, menjaga bentuk sel dan metastasis tumor (Amin, 2005)

2.2.5 Laminin

Laminin merupakan non-kolagen utama yang membentuk lapisan tipis pada matriks ektrasel (lamina basalis) dan membentuk lamina basalis bersama kolagen IV. Laminin berikatan dengan kolagen melalui entaktin dan perlekan. Lamina basalis merupakan lapisan tipis yang terdapat dibawah sel epitel dan mengelilingi sel otot, sel lemak dan sel Schwann (Amin, 2005).

Laminin sangat penting untuk melekatnya sel, diferensiasi dan pergerakan sel, mempertahankan bentuk, memperpanjang dan meningkatkan survival jaringan. Fungsi laminin ini berhubungan dengan sekuen asam amino spesifik. Sekuen polipeptida pada rantai alfa meningkatkan adhesi sel endotel. Saat ini telah diidentifikasi sebanyak 15 trimer laminin (Suki dkk, 2005) .

2.3 Peranan berbagai sel pada paru

2.3.1 Netrofil

Jumlah netrofil pada alveolus biasanya kurang dari 1%. Netrofil dapat meningkat menjadi 3-5 % karena kemotaktik faktor yang dihasilkan makrofag (Cantin dkk, 1985). Netrofil mensekresi berbagai enzim seperti

serine protease yang meliputi netrofil elastase, *cathepsin*, proteinase-3 dan matriks metaloproteinase (MMP). Protease yang dihasilkan netrofil adalah MMP-8 dan MMP-9 yang sangat berperan pada destruksi alveolar. Migrasi netrofil ke saluran napas dipengaruhi oleh *neutrofil chemotactic factor* (NCF) yang meliputi interleukin (IL)-8 dan lekotrien B4 (LTB4). Survival Netrofil dapat meningkat akibat pengaruh sitokin seperti *granulocyte-macrophage colony stimulating factor* (GM-CSF) dan *granulocyte colony stimulating factor* (G-CSF) (Barnes dkk, 2003).

2.3.2 Makrofag alveolar

Pada orang normal, makrofag alveolar merupakan pertahanan utama pada saluran napas bagian bawah. Jumlah makrofag alveolar pada alveolus mencapai 82% dari jumlah sel inflamasi (Cantin dkk, 1985). Makrofag mempunyai peran yang sangat penting dalam patofisiologi penyakit paru obstruktif kronik. Terjadi peningkatan jumlah makrofag 5-10 kali pada saluran napas, parenkim paru, cairan kumbah bronkus (BAL) dan sputum perokok.

Pada analisis morfometri didapatkan peningkatan makrofag pada jaringan dan rongga alveolar dapat mencapai 25 kali dibandingkan dengan perokok normal. Makrofag ini akan terlokalisir pada duktus dan dinding alveoli penderita emfisema. Terdapat korelasi antara jumlah makrofag pada saluran napas dengan beratnya PPOK (Barnes dkk, 2003). Makrofag alveolar diaktivasi oleh asap rokok dan menghasilkan mediator inflamasi seperti tumor nekrosis factor (TNF) alfa, IL-8, *CXC chemokines, monocyte chemotactic factor* (MCP)-1, LTB4 dan *reaktive oxygen species* (ROS). Makrofag alveolar juga mensekresi enzim elastolitik meliputi MMP-2, MMP-9, MMP-12, katepsin K,L,S dan netrofil elastase yang diambil dari netrofil. Makrofag alveolar pada penderita PPOK memproduksi lebih banyak protein inflamasi dan mempunyai aktivitas elastolitik yang lebih besar. Enzim elastolitik utama yang dihasilkan oleh makrofag alveolar adalah MMP-9 (Barnes dkk, 2003, Barnes, 2004).

2.3.4 Limfosit T

Terdapat peningkatan jumlah limfosit T pada parenkim paru, saluran napas perifer dan sentral pada penderita PPOK. Terdapat peningkatan limfosit CD-8+ dan CD-4+ tetapi peningkatan jumlah limfosit CD-4+ tidak terlalu tinggi sehingga perbandingan antara CD-4+ dan CD-8+ tetap lebih rendah (Barnes, 2004).

2.3.5 Eosinofil

Peranan eosinofil pada PPOK masih belum jelas. Beberapa penelitian menunjukkan peningkatan jumlah eosinofil yang inaktif pada saluran napas, lavase bronkus dan biopsi saluran napas penderita PPOK stabil. Eosinofil pada saluran napas diduga berhubungan dengan respons terhadap pengobatan steroid dan adanya penyakit penyerta seperti penyakit asma. Peningkatan jumlah eosinofil sputum yang lebih dari 3% menunjukkan adanya *Eosinophylic Bronchitis* yang merupakan bagian dari penyakit asma (Hargreave dkk, 1999; Barnes dkk, 2003; Barnes, 2004).

2.3.5 Sel dendrit

Sel ini mempunyai peran yang sentral pada inisiasi respons imun *innate* dan adaptif. Saluran napas dan parenkim paru mengandung sel dendrit yang terletak pada permukaan sehingga sel ini merupakan pemberi sinyal jika ada zat inhalan yang masuk kedalam paru. Sel dendrit juga dapat mengaktivasi mediator inflamasi dan sel imun seperti makrofag, netrofil dan limfosit-B, sehingga diduga sel ini mempunyai peranan yang penting dalam memberikan respons terhadap asap rokok dan zat inhalan lain. Terdapat peningkatan jumlah sel dendrit pada paru tikus yang terpapar oleh asap rokok. Sel dendrit memegang peranan penting

pada respons paru terhadap asap rokok dan inhalasi zat berbahaya sehingga sel denrit merupakan elemen kunci pada PPOK (Barnes dkk, 2003; Barnes, 2004).

2.6 Sel epitel

Sel epitel pada saluran napas merupakan barier fisiko-kimia yang kompleks dan sangat berperan dalam sistem pertahanan paru (Mills dkk, 1999). Epitel saluran napas dan alveoli diduga merupakan sumber penghasil dari mediator inflamasi dan protease. Akibat rangsangan oleh asap rokok, sel epitel memproduksi berbagai mediator seperti TNF-α, IL-1β, GM-CSF dan IL-8. Mediator-mediator ini menyebabkan terjadinya rekruitmen sel inflamasi terutama netrofil dan makrofag. Sel epitel ini juga merupakan sumber TGF-β yang dapat menginduksi fibrosis setempat pada saluran napas (Barnes, 2003; Barnes dkk, 2004).

3. Patogenesis PPOK type Emfisema

Terjadinya emfisema tidak disebabkan oleh faktor tunggal, tetapi disebabkan oleh multifaktor seperti gangguan pada elastase-antielastase, oksidan-antioksidan dan protease-antiprotease (Palilingan dkk, 1987; Senior dkk, 1999; Foronjy dkk, 2001).

3.1 Elastase - Antielastase

Laurel dan Erickson pada tahun 1965 mendapatkan adanya hubungan antara defisiensi AAT dengan terjadinya emfisema. Adanya gangguan keseimbangan antara elastase dan anti-elastase yang menyebabkan terjadinya emfisema. Tetapi banyak peneliti mempertanyakan teori ini, mengingat tidak semua penderita defisiensi AAT yang merokok akan menderita emfisema. Pada individu tanpa defisiensi AAT didapatkan bahwa kerusakan AAT disebabkan karena oksidan atau radikal bebas yang dapat menginaktivasi asam amino metionin pada posisi 358 (Donno dkk, 2000).

3.2 Oksidan- Antioksidan

Gangguan keseimbangan oksidan-antioksidan yang terjadi pada saluran napas bagian distal berperan pada patogenesis emfisema (Alsegaff dan Wijaya, 1992). Selain asap rokok, oksidan juga dapat dihasilkan oleh sel inflamasi. Oksidan ini akan mendegradasi matriks ekstrasel dan menginaktivasi antiprotease (Bridgeman dkk, 1991). Oksidan yang berhubungan dengan patogenesis timbulnya emfisema adalah (Palilingan dkk, 1987, Alsegaff dkk, 1992) :

1. Oksidan inhalan : Asap rokok,polutan, nitrogen dioksid dan ozon (O3)
2. Oksidan yang berasal dari sel radang : Anion superoksida, hidrogen peroksida, hidroksil, Singlet oksigen dan anion hipoklorit¬

3.2.1 Oksidan/Radikal bebas

Oksidan adalah bahan kimia elektrofil yang sangat reaktif, mempunyai kemampuan untuk memindahkan elektron dari berbagai molekul dan mengakibatkan terjadinya oksidasi pada molekul tersebut. Sebagai akseptor elektron, radikal bebas dapat mengubah struktur molekul protein dan lipid. Bila perubahan terjadi pada bagian yang penting dari molekul, akan menyebabkan molekul kehilangan fungsinya. Oksidan dapat merusak sel parenkim paru, alveolus dan matrik ekstrasel. Oksidan juga dapat diproduksi pada proses metabolisme normal dan sel radang.

Secara praktis, semua asam amino dapat menjadi target oksidatif oleh ROS tetapi asam amino yang lebih sensitif terhadap adalah triptofan, tirosin, histidin dan sistein. Akibat oksidasi terjadi perubahan atau modifikasi pada protein yang mengakibatkan terjadinya perubahan fungsi, fragmentasi kimia dan meningkatkan kepekaan terhadap serangan enzim proteolitik. Setelah terpapar superoksida atau hidrogen

peroksida terjadi peningkatan proteolisis sebanyak 11 kali (Droge, 2002). Oksidan sendiri kurang mampu merusak jaringan ikat pada parenkim paru, tetapi memerlukan enzim protease yang kuat seperti elastase. Gangguan keseimbangan oksidan dan antioksidan akan mengakibatkan oksidan dan elastase bekerja sama dalam menyebabkan kerusakan parenkim paru. Umumnya semua sel fagosit pada saat terjadinya fagositosis atau pada peradangan akut, di dalam sel terjadi proses *"respiratory burst"* yang ditandai dengan meningkatnya penggunaan oksigen dan disertai peningkatan produksi hidrogen peroksida dan penggunaan glukosa melalui *"shunt"* heksosa monofosfat.

Sel yang mengalami peristiwa tersebut adalah netrofil, monosit, makrofag dan eosinofil. Meningkatnya penggunaan oksigen saat *"respiratory burst"* diduga berhubungan dengan aktivitas enzim NAD(P)H yang mengakibatkan terbentuknya radikal anion superoksida (O2.-) :

$2O_2 + NAD(P)H \rightarrow 2O_2.- + NAD(P)^1 + H$

NAD(P) yang terbentuk akan merangsang *"shunt"* heksosa monofosfat, kemudian glukosa-6-fosfat diubah menjadi 6-fosfoglukonat oleh glukosa-6-fosfat dehidrogenase. Pada saat yang sama NAD(P) diubah kembali menjadi NAD(P)H. Asap rokok mengandung banyak radikal bebas yang dapat merusak dinding sel, sitosol dan *deoxyribo nucleic acid* (DNA). Radikal bebas pada asap rokok terdiri dari fase gas dan fase tar. Menurut Prior dan Stone (1993), fase gas asap rokok mengandung sekitar 1015 radikal bebas (terutama radikal alkil dan peroksil) pada setiap hisapan dan 500 – 1000 ppm nitrit oksida. Nitrit oksida ini akan segera bereaksi dengan anion superoksida (O_2.-) membentuk peroksinitrit (ONOO-) menghasilkan radikal hidroksil yang sangat toksis. Peroksinitrit kemudian dengan radikal peroksil akan membentuk alkil peroksil nitrit (ROONO). Fase tar asap rokok mengandung lebih dari 1018 radikal bebas per gram terdiri dari kompleks kuinon-hidrokuinon, radikal hidroksil, hidrogen peroksida dan *metal-chelator*. Kompleks kuinon-hidrokuinon bereaksi dengan oksigen membentuk molekul anion superoksida. *Metal-chelator* akan mengikat ion Fe membentuk tar-semikuinon dan tar Fe++ serta menghasilkan hidrogen peroksida (Aditama, 1995; MacNee dkk, 1999; Donno dkk, 2000; Barnes dkk, 2003).

Target utama radikal bebas pada dinding sel adalah *poly-unsaturated fatty acid* (PUFA). Oksidan atau radikal bebas menyebabkan terputusnya rantai asam lemak dan membentuk senyawa yang lebih toksik contohnya malondialdehid, 9-hidroksi nonenal dan bermacam-macam senyawa hidrokarbon yaitu etana (C2H6), pentana (C5H12). Oksidan atau radikal bebas juga menyebabkan peroksidasi asam arakhidonat menghasilkan F2-isoprostane (F2-iP) yang merupakan isomer dari prostaglandin (Suryohudoyo, 1995. Abuja dkk, 2001).

Peningkatan jumlah radikal bebas akibat paparan asap rokok yang terus menerus dapat mengakibatkan penurunan aktivitas antioksidan dan menyebabkan stres oksidatif. Tubuh mempunyai 2 jenis antioksidan yaitu antioksidan enzim dan non-enzim (Suyatna, 1995. Barnes, 2004). Antioksidan enzim yang utama pada paru adalah katalase, superoksid dismutase (SOD), glutation peroksidase (GPx). Yang termasuk antioksidan non-enzim adalah glutation, asam askorbat, asam urat, vitamin E, bilirubin dan asam lipoat. Aktivitas antioksidan intrasel relatif lebih rendah dibandingkan dengan ekstrasel dan tidak dipengaruhi oleh adanya stres oksidatif (Barnes, 2004).

Superoksid dismutase (SOD) mengubah anion superoksida yang terdapat pada asap rokok dan endogen menjadi hidrogen peroksida. Penurunan aktivitas SOD menyebabkan radikal anion superoksida tidak dapat diubah menjadi hidrogen peroksida. Glutation peroksidase (GPx) mengubah hidrogen peroksida menjadi air

(H2O) dengan bantuan glutation (GSH). Penurunan aktivitas glutation peroksidase menyebabkan hidrogen peroksida yang banyak terdapat pada asap rokok tidak dapat diubah menjadi H_2O.

Glutation (GSH) merupakan antiosidan yang memegang peranan penting pada ekstrasel, membran sel dan intrasel. Pada membrane sel GSH berperan mengembalikan dehidro askorbat (DHA) yang teroksidasi oleh radikal bebas menjadi asam askorbat. Pada ekstrasel hidrogen peroksida yang berasal dari asap rokok akan dinetralisir menjadi H2O oleh glutation peroksidase dengan bantuan GSH. Glutation intrasel akan menetralisir hidrogen peroksida yang masuk dari ekstrasel dan yang berasal dari endogen. Penurunan kadar GSH menyebabkan tidak semua hidrogen peroksida dapat diubah menjadi H2O oleh glutation peroksidase. Hidrogen peroksida dapat bereaksi dengan anion superoksida dengan bantuan logam transisi membentuk radikal hidroksil yang sangat toksik *(reaksi Haber-Weiss).*

Radikal hidroksil yang sangat toksik ini akan dinetralisir oleh senyawa yang mengandung gugus sulfhidfril seperti glutation (GSH) dan sistein (Cys-SH) (Suryohudoyo, 2007). Hidrogen peroksida masuk kedalam sel, merusak ikatan IkB dengan NFkB. *Nuclear-factor* kappa B (NFkB yang bebas akan memasuki nukleus dan merangsang produksi faktor pro-inflamasi dan menyebabkan rekruitmen sel inflamasi terutama makrofag dan netrofil. Makrofag dan netrofil memproduksi enzim protease termasuk matriks metaloproteinase. Diantara matriks metaloproteinase, MMP-8 dan MMP-9 berperan merusak matriks ekstrasel (MES) terutama elastin sehingga menyebabkan paru kehilangan elastisitasnya. Sangat penting untuk menetralisir anion superoksida dan hidrogen peroksida untuk mencegah terbentuknya radikal hidroksil yang sangat toksik *(reaksi Haber-Weiss)* dan rekruitmen sel inflamasi yang menghasilkan MMP-8 dan MMP-9.

Sistem glutation merupakan antioksidan yang penting pada saluran napas dan kadarnya meningkat pada perokok dan stres oksidatif (Barnes, 2004). Mengingat pentingnya peran GSH untuk menetralisir hidrogen peroksida dan radikal hidroksil, penurunan kadar GSH merupakan faktor yang sangat menentukan kerentanan sel terhadap oksidan. Kadar GSH yang rendah pada cairan permukaan epitel paru dapat menyebabkan kematian sel dan kerusakan paru (Suyatna, 1995; Donno dkk, 2000). Kerjasama oksidan terutama radikal hidroksil dan matriks metaloproteinase ini menyebabkan kerusakan pada MES terutama elastin sehingga menimbulkan emfisema. Kerusakan elastin menyebabkan paru kehilangan elastisitas *(loss of elasticity).*

Asap rokok merupakan campuran gas-gas dan bahan partikel. Campuran ini mengandung lebih dari 4700 bahan kimia terutama oksidan atau radikal bebas, sekitar 1000 adalah berbentuk gas (Aditama, 1995; MacNee, 1995; Donno dkk, 2000). Setiap hembusan asap rokok mengandung 1015 radikal bebas (terutama alkil dan peroksil) dan 800 ppm nitrogen oksida (NOx) yang dengan cepat akan bereaksi dengan superoksida anion membentuk peroksinitrit.

Fase tar mengandung 1018 radikal bebas/gram yang terdiri dari radikal hidroksil, hidrogen peroksida, kompleks kuinon-hidrokuinon dan *metal chelator.* Kompleks kuinon-hidrokuinon beraksi dengan oksigen dan membentuk anion superoksida (Donno dkk, 2000).

Asap rokok dapat langsung mempunyai efek toksis terhadap jaringan paru dan dapat menginaktivasi enzim yang melindungi paru terhadap efek elastase yang dihasilkan oleh netrofil. Oksidan yang masuk saluran napas terutama berasal dari asap rokok (eksogen) dan endogen, akan diredam oleh antioksidan (radical scavenger) yang terdapat pada saluran napas. Anion superoksida akan bereaksi dengan hidrogen peroksida dengan bantuan ion Fe++ membentuk radikal hidroksil (OH-) yang sangat reaktif (reaksi *Fenton*

dan *Haber-Weis*) dan dapat merusak semua jaringan (MacNee dan Rahman, 1999). Anion superoksida juga dapat beraksi dengan nitrit oksida membentuk peroksinitrit dan menghasilkan radikal hidroksil (Barnes,2004).

Pada pernapasan fisiologis juga terbentuk 4–5% radikal bebas. Apabila jumlah radikal bebas yang dihasilkan berlebihan maka dapat menyebabkan terjadinya gangguan keseimbangan antara oksidan-antioksidan (stres oksidatif). Stres oksidatif ini dapat menyebabkan rusaknya struktur sel dan perubahan patologi. Radikal bebas adalah fragmen molekul kecil yang berperan secara langsung dalam aktifitas metabolik sel. Pada proses yang patologis seperti inflamasi jaringan, terjadi pembentukan radikal bebas yang berlebihan dan dapat menyebabkan kerusakan yang irreversibel dan kematian sel (Hermawan, 2011. Suyatna, 1995). Radikal bebas merupakan atom atau molekul yang mengandung sejumlah elektron yang cacat, elektron di orbit terluar tidak berpasangan. Radikal bebas sangat reaktif, karena memiliki sejumlah elektron yang tidak berpasangan dan cenderung untuk mengambil elektron dari molekul lain atau dengan melepas elektronnya sendiri.

Target utama yang dirusak oleh radikal bebas adalah asam lemak *poli-unsaturated fatty acid* dan asam lemak pada dinding sel. Kerusakan ini menyebabkan terbentuknya peroksida lemak dan jika reaksi terus berlanjut akan menghasilkan peroksida dan aldehid. Jumlah produksi peroksida lipid diukur sebagai TBARS (thiobarituric acid-reaktif substances). Peroksida lipid ini akan mengalami transformasi dan membentuk radikal-radikal lain. Derivat oksigen yang disebut jenis oksigen reaktif (ROS) bekerja sebagai oksidan utama. Sekali diproduksi, metabolik reaktif ini dapat merubah struktur selular, metabolisme sel, berintegrasi dengan asam lemak tak jenuh dan asam nukleat sehingga menyebabkan perubahan pada DNA seperti mutasi.

3.2.1.1 Produksi Radikal Bebas

Secara normal radikal bebas terdapat dalam sistem biologi dan sangat penting untuk mempertahankan hidup. Sumber radikal bebas dalam tubuh berasal dari metabolisme aerobik dan spesies molekul oksidatif (Makmun, 2005) ;

1. Proses biokimia dalam tubuh
   Pernapasan aerob, reduksi molekul oksigen menghasilkan superoksida danradikal hidroksil. Hasil sampingan proses oksidasi katekolamin, aktivasi kaskade asam arakidonat dan produksi HOCl dari H_2O_2 (fagosit).
2. Radiasi elektromagnetik, seperti sinar gamma ; $H_2O \rightarrow OH. + e + H^+$
3. Asap rokok, Pada setiap hembusan mengandung 1015 radikal bebas
   3.1 $NO \rightarrow NO_2 \rightarrow RO \rightarrow ROO \rightarrow RO + NO_2$
   3.2 $NO + H_2O_2 \rightarrow HNO_2 + OH.$
   3.3 $NO_2 + H_2O_2 \rightarrow HNO_3 + OH.$ (OH. merusak elastin di paru)
4. Polutan di udara ; NO_2, O_3, SO_2
5. Akibat proses peradangan (inflamasi).

Inflamasi oleh sel granulosit, monosit dan makrofag dapat menghasilkan oksidan seperti H_2O_2, OH., HClO- dan O_2.-. Jika terjadi peradangan hebat maka produksi oksidan akan meningkat (respiratory burst) dan dapat menghancurkan mikroorganisme dan menyebabkan kerusakan pada jaringan.

3.2.1.2 Dampak oksidan terhadap inflamasi

Asap rokok dapat menyebabkan iritasi pada sel epitel saluran napas dan menimbulkan reaksi inflamasi. Reaksi inflamasi terjadi pada saluran napas utama, saluran napas perifer dan parenkim paru (Shapiro, 1999. Saetta, 1999). Penyakit paru obstruktif kronik merupakan penyakit inflamasi yang kompleks dan melibatkan berbagai sel serta mediator inflamasi (Barnes dk, 2003).

Radikal bebas atau *reaktif oxygen species* (ROS) menyebabkan degradasi *Inhibitory kappa-B* (IkB) sehingga terjadi aktivasi nuclear factor kappa-B (NFkB). NFkB yang bebas akan memasuki nukleus dan merangsang produksi faktor inflamasi sehingga terjadi rekruitmen netrofil dan makrofag alveolar. Netrofil dan makrofag alveolar memproduksi elastase, kolagenase yang dapat menyebabkan terjadinya kerusakan pada matriks ekstrasel (MES). Radikal bebas menyebabkan kerusakan atau disfungsi sel yang diikuti oleh reaksi inflamasi dan perubahan degeneratif.

Inflamasi pada emfisema merupakan proses yang multipel (Amin, 2005) :

1. Asap rokok dapat merekrut sel inflamasi secara langsung
2. Asap rokok menginduksi sel epitel untuk melepaskan senyawa pro-inflamasi
3. Terjadinya infeksi dapat menimbulkan reaksi inflamasi sehingga memperberat inflamasi yang sudah ada.

Radikal bebas juga menyebabkan rekruitmen berbagai sel inflamasi terutama netrofil dan makrofag (Barnes, 2003). Pada konsentrasi sedang, nitrit oksida (NO), anion superoksida dan ROS mempunyai peran yang penting sebagai mediator regulasi pada proses sinyal yang beberapa diantaranya berperan memproteksi sel dari serangan oksidatif. Pada konsentrasi tinggi, radikal bebas, derivat radikal dan non radikal reaktif spesies berbahaya terhadap organisme hidup dan merusak semua komponen utama sel (Droge, 2002)

3.2.1.3 Dampak oksidan terhadap netrofil

Asap rokok menyebabkan peningkatan jumlah netrofil pada saluran napas menjadi 3 – 5% (Cantin dkk, 1985). Migrasi netrofil ke saluran napas dipengaruhi oleh *neutrophyl chemotactic factor* (NCF) yang meliputi interleukin-8 (IL-8) dan lekotrien B4 (LTB4). Survival netrofil ini meningkat akibat pengaruh sitokin seperti *granulocyte-macrophage colony stimulating factor* (GM-CSF) dan granulocyte colony stimulating factor (G-CSF) (Barnes dkk, 2003). Enzim elastolitik utama yang dihasilkan netrofil adalah MMP-8 dan MMP-9. MMP-8 dan MMP-9 tidak hanya berfungsi sebagai enzim yang disekresi tetapi juga melekat pada sel dimana terjadi aktivitas elastolitik. Sekitar 80% MMP-8 dan MMP-9 yang dihasilkan netrofil tetap berhubungan dengan permukaan netrofil, dinetralisir oleh TIMPs sehingga memegang peranan penting dalam elastolisis. Terdapat peningkatan yang bermakna aktivitas MMP-8 dan MMP-9 pada perokok yang menderita PPOK dibandingkan dengan perokok yang tidak menderita PPOK (Vernooy dkk, 2004)

3.2.1.4 Dampak oksidan terhadap makrofag alveolar

Makrofag mempunyai peran yang sangat penting dalam patofisiologi penyakit paru obstruktif kronik. Terdapat peningkatan 5-10 kali jumlah makrofag alveolar pada saluran napas, parenkim paru, cairan BAL dan sputum perokok (Barnes, 2000). Makrofag alveolar diaktivasi oleh asap rokok dan menghasilkan mediator inflamasi seperti tumor nekrosis factor (TNF) alfa, IL-8, CXC *chemokines, monocyte chemotactic factor* (MCP)-1, LTB4 dan *reaktif oxygen species* (ROS). Makrofag alveolar juga mensekresi enzim elastolitik meliputi MMP-2, MMP-9, MMP-12, cathepsin K,L,S dan netrofil elastase yang diambil dari netrofil.

Makrofag alveolar pada penderita emfisema memproduksi lebih banyak protein inflamasi dan mempunyai aktivitas elastolitik yang lebih besar. Enzim elastolitik utama yang dihasilkan oleh makrofag alveolar penderita PPOK adalah MMP-9 (Barnes dkk, 2003).

Makrofag alveolar juga merupakan sumber dari ion Fe pada paru. Pada cairan *broncho-alveolar lavage* (BAL), 80% ion Fe terutama terdapat didalam sel makrofag alveolar dan 20% terdapat pada cairan epitelial. Didalam makrofag 25% ion Fe terikat dengan ferritin sedangkan 75% ditemukan pada mitokondria dan lisosom. Ion Fe mempunyai sifat yang reaktif dan terikat dengan transferin, seruloplasmin dan ferritin. Pada perokok didapatkan jumlah ion Fe meningkat pada makrofag alveolar dan cairan epitelial. Makrofag alveolar pada perokok menghasilkan lebih banyak ion Fe dibandingkan bukan perokok (Donno, 2000).

3.2.1.5 Dampak negatif oksidan

Dari berbagai senyawa oksigen reaktif, radikal hidroksil merupakan senyawa yang paling berbahaya karena mempunyai sifat yang sangat reaktif (Makmun, 2005; Suryohudoyo, 2007)

3.2.1.5.1 Dampak negatif oksidan terhadap membran sel

Semua komponen sel rentan terhadap serangan radikal bebas, tetapi sistem membran adalah target utama. Kerentanan membran sel disebabkan karena kandungan asal lemak yang tinggi. Komponen terpenting dari membran sel adalah fosfolipid, glikolipid dan kolesterol. Fosfolipid dan glikolipid mengandung asam lemak jenuh (asam linoleat, linolenat dan arakidonat) yang sangat rentan terhadap serangan radikal bebas, terutama radikal hidroksil. Kerentanan membran sel bagian dalam disebabkan adanya asam lemak PUFA. Semakin tinggi tingkat tak jenuh dari asam lemak, semakin rentan terhadap serangan radikal bebas.

Radikal hidroksil (OH.) dapat menyebabkan reaksi berantai yang disebut peroksidasi lipid (Makmun, 2002).

LH	+	OH^-	$\rightarrow$	L.	+	H2O	radikal lipid
L	+	O2	$\rightarrow$	LOO.			radikal peroksilipid
LOO	+	LH	$\rightarrow$	L.	+	LOOH	radikal lipid

Hasil akhir dari reaksi menyebabkan terputusnya rantai asam lemak menjadi senyawa yang bersifat toksik terhadap sel yaitu aldehid seperti malondialdehid (MDA), 8-hidroksi-nonenal serta berbagai senyawa hidrokarbon seperti etana (C_2H_6), pentana (C_5H_{12}). Dapat juga terjadi reaksi silang antara dua rantai asam lemak atau antara asam lemak dengan rantai peptida (protein) yang timbul karena reaksi dua radikal.

R1. + R2. $\rightarrow$ R1-R2

Akibat dari reaksi ini dapat menyebabkan kerusakan yang parah pada membran sel dan membahayakan kehidupan sel (Suryohudoyo, 1995).

3.2.1.5.2 Dampak negatif oksidan terhadap DNA

Radikal bebas dapat menimbulkan berbagai perubahan pada DNA berupa hidroksilasi basa timin dan sitosin serta guanosin menjadi *8-hidroxy-2 deoxiguanosin*, pembukaan inti purin dan pirimidin serta terputusnya rantai fosfodiester DNA.

Bila kerusakan tidak terlalu parah, maka masih dapat diperbaiki oleh sistem perbaikan DNA *(DNA repair*

system) yaitu *DNA glucosylase.* Tetapi jika kerusakan terlalu parah, misalnya DNA terputus-putus pada berbagai tempat maka kerusakan tersebut tidak dapat diperbaiki sehingga menyebabkan gangguan replikasi sel.

Perbaikan DNA ini justru sering menimbulkan mutasi akibat sistem perbaikan DNA cenderung membuat kesalahan *(error prone)* yang apabila mengenai gen tertentu dapat terjadi mutasi dapat menimbulkan kanker (Suryohudoyo, 1995).

3.2.1.5.3 Dampak negatif oksidan terhadap protein

Oksidan dapat merusak protein dengan bereaksi terhadap asam amino yang menyusun protein. Secara praktis, semua asam amino dapat menjadi target oksidatif dari ROS tetapi yang sensitif terhadap ROS adalah asam amino seperti triptofan, tirosin, histidin dan sistein. Dari berbagai asam amino penyusun protein, asam amino yang paling sensitif adalah sistein.

Modifikasi oksidatif akibat ROS menyebabkan perubahan fungsi, fragmentasi kimia dan meningkatkan kepekaan terhadap enzim proteolitik. Kepekaan terhadap enzim proteolisis meningkat 11 kali setelah terpapar superoksida anion atau hidrogen peroksida (Droge, 2002).

Sistein yang terdapat pada senyawa sulfhidril (-SH) yang justru paling peka terhadap serangan radikal bebas seperti radikal hidroksil ;

$$RSH + OH^{\cdot} \rightarrow RS^{\cdot} + H2O$$
$$RS. + RS^{\cdot} \rightarrow R\text{-}S\text{-}S\text{-}R$$

Pembentukan ikatan disulfide (-S-S-) menimbulkan ikatan intra dan antar molekul menyebabkan protein kehilangan fungsi biologisnya (enzim kehilangan aktivitas) (Suryohudoyo, 2007).

3.2.1.5.4 Dampak positif oksidan

Oksidan dapat menimbulkan dampak yang merugikan, tetapi dampak ini juga dapat dimanfaatkan oleh tubuh untuk melawan orgasnisme patogen. Untuk menghadapi organisme patogen ini tubuh mempunyai sel yang khusus yaitu granulosit, monosit dan makrofag yang dapat menghasilkan oksidan seperti H_2O_2, $OH^{\cdot}$, $HClO^{-}$, $O_2^{-\cdot}$dan singlet oksigen. Tetapi perlu diingat bahwa oksidan ini selain dapat menghancurkan mikroorganisme juga dapat merusak sel-sel jaringan tubuh sendiri, menyebabkan terjadi peradangan hebat yang melibatkan banyak sel radang dan menimbulkan kerusakan pada jaringan (Suryohudoyo, 1995).

3.2.2 Antioksidan

Antioksidan adalah senyawa yang dalam kadar rendah dibanding bahan yang dapat dioksidasi, sangat memperlambat atau menghambat oksidasi bahan tersebut. Tubuh manusia juga telah menyiapkan antioksidan sebagai pertahanan untuk menghadapi serangan radikal bebas ditingkat sel, membran dan ektrasel (Suryohudoyo, 1995). Antioksidan dapat mengubah molekul oksidan menjadi molekul yang tidak berbahaya atau menjadi molekul yang tidak dapat mempengaruhi jaringan.

Pada keadaan normal antioksidan dapat mencegah terjadinya kerusakan jaringan akibat pengaruh oksidan. Antioksidan berfungsi untuk mempertahankan saluran napas terhadap pengaruh oksidan. Antioksidan terdapat pada intrasel, membran sel maupun ruang ekstrasel. Antioksidan intraseluler utama pada paru

adalah katalase, superoksid dismutase dan sistem glutation. Antioksidan mengubah oksidan menjadi molekul yang tidak berbahaya atau menjadi molekul yang tidak dapat mempengaruhi jaringan vital.
Pertahanan antioksidan terdiri dari :

1. Bahan kimia yang dapat mengkatalisis radikal bebas dan spesies reaktif misalnya SOD, katalase, peroksidase dan antioksidan tiol spesifik.
2. Protein yang meminimalisir pro-oksidan seperti ion Fe, Cu dan haem.
3. Protein yang memproteksi biomolekul, mencegah kerusakan dengan mekanisme lain
4. Bahan kimia dengan berat molekul rendah yang dapat menetralisir spesies oksigen reaktif dan spesies nitrogen reaktif.

Antioksidan seperti N-asetil sistein (NAC) dapat menghambat aktivasi NFkB dan degradasi IKb2 (Droge, 2002)

3.2.2.1 Mekanisme Kerja Antioksidan

Jika sistem pertahanan antioksidan berfungsi dengan baik, sebagian besar komponennya berada dalam bentuk tereduksi *(reduced)* H_2O_2, GSH dan NADPH. Tetapi jika terjadi malfungsi maka cenderung terjadi akumulasi H_2O_2, GSSG dan NADP. Untuk meredam dampak negatif oksidan dapat dilakukan dengan 2 cara yaitu dengan mencegah terbentuknya senyawa oksidan secara berlebihan dan mencegah berlanjutnya reaksi berantai.

Berdasarkan mekanisme pencegahan terhadap dampak negatif oksidan, maka antioksidan dapat dibagi menjadi 2 golongan (Suryohudoyo,1995) ;

1. Antioksidan pencegah *(preventive anti-oxidants)*
2. Antioksidan pemutus rantai *(chain-breaking anti-oxidants).*

3.2.2.1.1 Antioksidan Pencegah

Pada prinsipnya antioksidan ini bertujuan untuk mencegah terbentuknya radikal hidroksil yang merupakan radikal yang sangat berbahaya. Ada 3 komponen yang diperlukan untuk membentuk radikal hidroksil yaitu logam transisi Fe atau Cu, H_2O_2 dan O_2.-. Untuk menghambat reaksi Fenton, maka harus dicegah keberadaan ion Fe++ oleh transferin atau feritin dan ion Cu+ oleh seruloplasmin dan albumin. Penimbunan O_2.- dapat dicegah oleh enzim superoksid dismutase (SOD) sedangkan penimbunan H_2O_2 dapat dicegah oleh katalase dan peroksidase. Salah satu enzim peroksidase yang paling penting adalah glutation peroksidase (Suryohudoyo, 1995)

3.2.2.1.2 Antioksidan Pemutus Rantai

Antioksidan yang termasuk dalam kelompok ini adalah vitamin E (tocoferol), asam ascorbat, beta karoten dan 2 senyawa yang berperan sebagai pencegah yaitu glutation dan sistein. Vitamin E dan beta karoten bersifat lipofilik dan berperan pada membran sel untuk mencegah peroksidasi lipid, sedangkan vitamin C, sistein dan glutation bersifat hidrofilik dan berperan pada sitosol.

Vitamin E bereaksi dengan radikal lipid dan radikal peroksilipid membentuk radikal vitamin E yang tak terlalu reaktif karena terjadi resonansi dan dapat dihilangkan melalui reaksi intramolekul, bergerak kepermukaan dan bereaksi dengan vitamin C dan kemudian dihilangkan melalui reaksi dismutasi. Vitamin E hanya dapat berperan pada tekanan oksigen (PaO_2) tinggi, sehingga bila tekanan O_2 rendah peranan vitamin E digantikan oleh beta karoten yang sangat stabil karena adanya resonansi pada molekulnya (Suryohudoyo, 1995).

Komposisi pertahanan antioksidan berbeda pada tiap jaringan dan sel yang satu dengan sel yang lainnya. Pertahanan antioksidan dapat di induksi oleh paparan terhadap spesies oksigen reaktif atau spesies nitrogen reaktif dan sitokin. Para ahli mengetahui bahwa pertahanan antioksidan ini tidak sepenuhnya komplit karena tidak dapat mencegah kerusakan secara menyeluruh seperti kerusakan DNA, protein, lipid dan molekul kecil yang terjadi pada sistem kehidupan dengan udara biasa (Halliwell dkk, 1999).

Antioksidan yang berperan pada jalan napas bagian bawah (Cantin dkk, 1985; Palilingan dkk, 1987) :

1. Antioksidan intrasel :
   a. Superoksid dismutase.
   b. Katalase.
   c. Sistem glutation.
2. Antioksidan membran sel ;
   Vitamin E
3. Antioksidan ekstrasel.
   a. Katalase.
   b. Glutation
   c. Seruloplasmin.
   d. Alfa1 antitripsin
   e. Vitamin A
   f. Metionin.
   g. Bilirubin.

Pengobatan dengan pemberian antioksidan seperti N-asetil sistein (NAC) dapat mencegah aktivasi NFkb dan degradasi Ikb2. Ada 2 mekanisme yang dapat meningkatkan aktivasi NFkb yaitu meningkatnya degradasi ikatan NFkb dengan Ikb dan meningkatkan aktivitas enzim Ikb2 kinase (Droge, 2002)

3.2.2.1.1 Superoksid Dismutase (SOD)

Superoksid dismutase mempunyai berat molekul 32.000, mempunyai 2 sub-unit dan salah satu sub-unit mengandung Cu yang aktif pada proses katalisis, sedangkan sub-unit yang lain mengandung Zn yang tidak berfungsi pada katalisis tetapi berfungsi untuk stabilitas enzim. Superoksid dismutase (SOD) mempunyai 3 iso-enzim yaitu Manganese-SOD (mitokhondria), Cu-Zn-SOD (mitokhondria), Cu-Zn-SOD (MES). Terdapat 2 bentuk SOD yaitu yang mengandung Cu-Zn (sitoplasma) dan Mg-Zn (mitokondria) (Cantin dkk.1985).

Masih terdapat perdebatan tentang fungsi superoksid dismutase sebagai antioksidan sesungguhnya mengingat bahwa SOD mengubah anion superoksida yang tidak begitu kuat menjadi hidrogen peroksida yang merupakan oksidan yang lebih kuat (Barnes dkk, 2003). Penelitian yang dilakukan Kluchova dkk (2007) pada PPOK sedang dan berat didapatkan peningkatan bermakna aktivitas SOD pada PPOK berat (1039,3 = 20,8 vs 997,4 = 34,6)U/gHb. Superoksid dismutase ini diproduksi oleh sel inflamasi (Cantin dkk, 1985).

$$\text{Enzim-Cu}^{2+} + O_2^{\cdot -} \rightarrow \text{Enzim-C}^{u+} + O_2$$

$$\text{Enzim-Cu}^{+} + O_2^{\cdot -} + 2H^{+} \rightarrow \text{Enzim-Cu}^{2+} + H2O2$$

Hasil akhir $O_2^{\cdot -} + O_2^{\cdot -} + 2H^{+} \rightarrow H_2O_2 + O_2$

Anion superoksida dapat mengubah ion ferri (Fe3+) menjadi ion ferro (Fe2+) yang dapat memberikan satu elektron pada H2O2 sehingga terbentuk radikal hidroksil (OH.) yang sangat toksis terhadap jaringan.

$Fe^{3+} + O2^{\cdot-} \rightarrow Fe^{2+} + O2$

$Fe^{2+} + H_2O_2 \rightarrow OH^- + OH^{\cdot} + Fe^{3+}$

atau

$Cu^{2+} + O_2^{\cdot-} \rightarrow Cu^{+} + O_2$

$Cu^{+} + H_2O_2 \rightarrow Cu^{2+} + OH^- + OH^{\cdot}$

Hasil akhir $O_2^{\cdot-} + H_2O_2 \rightarrow O_2 + OH^- + OH^{\cdot}$

Reaksi radikal superoksida dengan hidrogen peroksida menghasilkan radikal hidroksil dan dikatalisis oleh ion metal disebut reaksi Haber-Weiss. Reaksi ini dapat dicegah oleh katalase yang dengan cepat mengubah H2O2 menjadi H2O. Katalase berfungsi sebagai katalisator pada reaksi H2O2 menjadi H2O dan O2 sehingga mencegah H2O2 menjadi radikal yang lebih toksis seperti radikal hidroksil dan anion hipohalida (Cantin dkk.1985; Halliwell dkk; 1999; Donno, 2000).

3.2.2.1.1.3 Glutation tereduksi (GSH)

Glutation merupakan suatu tripeptida yang terdiri dari glutamin, glisin dan sistein. Glutation mempunyai efek antioksidan karena glutation mengandung sistein yang menjadi sumber sulfhidril. Glutation merupakan antioksidan utama pada saluran napas. Kadar GSH yang tinggi terdapat pada cairan permukaan epitelial dan kadar ini semakin meningkat pada perokok (Barnes dkk, 2003). Glutation mampu melakukan detoksikasi terhadap hidrogen peroksida, lipid peroksida dan peroksida lainnya. Dalam proses ini *"reduced glutathione"* (GSH) mengalami oksidasi menjadi *oxidized glutathione* (GSSG) yang merupakan gabungan dua molekul glutation yang dihubungkan dengan gugus disulfida oleh enzim glutation peroksidase. Untuk mengubah GSSG menjadi GSH diperlukan enzim glutation reduktase dengan bantuan NADPH.

Proses regenerasi ini memerlukan hormon tiroid, fungsi mitokhondria dan metabolisme pentosa posfat. Untuk metabolisme pentosa posfat diperlukan enzim *"shunt"* heksosa monofosfat melalui reaksi NAD(P)H. Untuk mengubah NAD(P) menjadi NAD(P)H diperlukan enzim glukosa-6-fosfat dehidrogenase yang akan mereduksi NAD(P) menjadi NAD(P)H. Aktivitas *"shunt"* heksosa monofosfat meningkat pada fagositosis dan keadaan ini merupakan suatu mekanisme untuk mengeluarkan kelebihan H_2O_2.

Glutation intrasel yang terdapat dalam bentuk teroksidasi (GSSG) hanya kurang dari 1%, dari total glutation. Glutation sangat efisien dalam reaksi-reaksi biokimia dan selalu dipakai untuk menghadapi oksidan (Cantin dkk, 1985). Glutation (GSH) merupakan antioksidan yang sangat penting pada sistem pertahanan paru dan mempertahankan integritas dinding sel akibat inflamasi dan inhalasi oksidan atau radikal bebas endogen. Kadar GSH yang rendah pada cairan epitelial paru dapat menyebabkan meningkatnya inflamasi dan kerusakan paru. Kadar GSH pada cairan epitel paru dapat mencapai 100 x lebih tinggi dibandingkan plasma (Suyatna, 1995; Donno dkk, 2000).

Kadar GSH meningkat pada perokok kronik tetapi tidak pada perokok akut. Penelitian Cavarra dkk (2001) didapatkan penurunan kadar glutation total sesudah merokok dibandingan sebelum merokok (17,38 ± 2.61 vs 19,44 ± 3.53) µM sedangkan kadar GSSG sesudah merokok lebih tinggi dibandingkan sebelum merokok (0.73 ± 0,05 vs 0,54 ± 0,03) µM. Rasio GSH/GSSG dapat memberikan gambaran yang penting terhadap keseimbangan antara oksidan dan antioksidan (van der Vaart, 2004).

3.2.2.1.1,4 Glutation Peroksidase

Enzim ini pertama kali ditemukan tahun 1957 oleh Gordon C. Mill dengan glutation sebagai donor hidrogen. Glutation peroksidase (GPx) merupakan tetramer glikoprotein yang mengandung selenium dengan 4 asam amino seleno-sistein. Struktur glutation peroksidase terdiri dari 4 sub-unit glikoprotein (tetramer).

Bagian aktif glutation peroksidase mengandung atom selenium sebagai *selenocystein* dimana atom sulfur digantikan oleh atom selenium (Halliwell, 2004).

Mekanisme kerja GPx terdapat pada seleno-sistein dimana bentuk Se(-) berada dalam keadaan istirahat. Kemudian dioksidasi oleh enzim peroksidase menjadi Se-SG. Glutation (GSH) akan mengubah kembali menjadi Se(-) dengan membentuk GSSG. Integritas membran seluler dan subseluler sangat tergantung pada enzim ini. Aktivitas antioksidan intraseluler pada paru relatif lebih rendah karena tidak dipengaruhi oleh stres oksidatif. Inaktivasi hidrogen peroksida oleh enzim glutation peroksidase dengan bantuan GSH.

$$H_2O_2 + 2\ GSH \rightarrow GSSG + 2H_2O$$

Glutation peroksidase (GPx) merupakan antioksidan ekstrasel yang penting pada paru. Glutation peroksidase mereduksi lipid hidroperoksida menjadi alkohol dan mereduksi hidrogen peroksida menjadi H2O. Antioksidan ini dihasilkan oleh sel epitelial paru dan makrofag alveolar terutama pada respons terhadap asap rokok dan stres oksidatif. Glutation peroksidase ini dapat menginaktivasi hidrogen peroksida, anion superoksida dan menghalangi spesies nitrogen reaktif (Barnes dkk, 2003). Penelitian yang dilakukan Kluchova dkk (2007) didapatkan aktivitas GPx yang lebih rendah bermakna pada PPOK berat dibandingkan PPOK sedang (47,7 ± 2,9 vs 43,1 ± 1,5) U/gHb. Penelitian yang dilakukan Trachova dkk (2007) didapatkan penurunan bermakna aktivitas GPx pada PPOK dengan eksaserbasi akut.

3.2.2.1.1.5 Vitamin E

Vitamin E, vitamin C dan betakaroten adalah antioksidan yang melindungi paru dari kerusakan oksidatif oleh asap rokok dan polusi. Vitamin E terdapat pada cairan ekstrasel paru dan membran sel yang berfungsi untuk mengubah radikal oksigen dan radikal peroksil lipid menjadi bentuk yang kurang reaktif (Smit, 2001). Pada binatang dengan defisiensi vitamin E cenderung sensitif terhadap kerusakan jaringan paru apabila terpapar oleh oksidan.

Penelitian yang dilakukan Cavarra dkk (2001) didapatkan peningkatan kadar vitamin E sesudah merokok dibandingkan sebelum merokok (8,12 ± 2,45 vs 5,62 ± 2,30). Pemberian vitamin E dapat menghambat oksidan inhalan seperti ozon, NO_2 dan NO. Sel memiliki vitamin E pada permukaan luar membran dan dapat mencegah oksidasi lipid dengan memberikan ion H pada radikal tersebut sehingga dapat mencegah reaksi berikutnya. Vitamin E berperan dalam menghentikan rantai reaksi peroksidasi lipid oleh peroksida. Vitamin E dan beta-karoten juga mempunyai efek protektif terhadap gejala respirasi dan faal paru (Smit,2001)

3.2.2.1.1.6 Vitamin C

Efek antioksidan vitamin C sudah dikenal baik. Vitamin C adalah *radical-scavenger* yang terdapat pada cairan ekstrasel dan intrasel yang dapat menetralisir radikal bebas secara langsung pada cairan ekstrasel dan bertindak secara tak langsung dengan mengaktifkan vitamin E. Vitamin C dan E bekerja sinergis seperti sebuah konsert.

Vitamin C bersifat hidrofilik dan dapat mengurangi kerusakan paru oleh ozon atau oksigen hiperbarik.

Vitamin C banyak terdapat dalam cairan yang melapisi alveoli sehingga berfungsi sebagai antioksidan ekstrasel. Penelitian Cavarra dkk (2001) didapatkan penurunan kadar vitamin C sesudah merokok dibandingkan sebelum merokok (80,1 ± 9,5 vs 144,7 ± 10,1).

3.2.2.1.1.7 Vitamin A

Beta-karoten merupakan *radical scavenger* yang bersifat lipofilik dan berperan untuk mencegah peroksidasi lipid. Beta-karoten bersama vitamin E dan C melindungi paru dari kerusakan oksidatif. Vitamin A, metionin dan bilirubin dapat menginaktivasi anion seperti halida atau singlet oksigen (Smit, 2001; Suryohudoyo, 2007).

3.2.2.1.1.8 Seruloplasmin

Seruloplasmin adalah antioksidan plasma yang memberi perlindungan antioksidan pada ruang ekstrasel, dinding alveoli. Seruloplasmin berfungsi mempertahankan ion Fe dalam bentuk ion feri (Fe^{+++}) sehingga dapat menurunkan jumlah ion Fe^{++}. Dengan demikian secara tidak langsung seruloplasmin dapat mencegah perubahan H2O2 menjadi radikal hidroksil yang toksik. Seruloplasmin juga dapat mencegah peroksidasi lemak dan mencegah oksidasi alfa-l-antitripsin.

3.3.6 Pertahanan sel

Tubuh manusia dilengkapi dengan pertahanan berupa antioksidan untuk menangkal serangan radikal bebas baik intrasel, membran sel dan ekstrasel (Makmun, 2002).

3.3.6.1 Pertahanan intrasel

Terdapat kerjasama antara superoksid dismutase, peroksidase dan katalase. Produksi radikal bebas intrasel sangat berkurang karena oksidase sitokrom pada mitokondria membantu transport elektron tanpa menghasilkan ROS.

$2O_2^{\cdot-} + 2H \rightarrow H_2O_2 + O_2$ dengan bantuan SOD sitosol dan mitokondria,
Cu, Zn dan Mn. H_2O_2 mudah melewati membran sel.
$Fe^{2+} + H_2O_2 \rightarrow Fe^{3+} + OH^{\cdot} + OH.$ (Reaksi Fenton)
$2GSH + H2O2 \rightarrow GSSG + 2H_2O^{\cdot}$ (dalam sitosol dan mitokondria)
$2H_2O_2 \rightarrow 2H_2O + O_2$

3.3.6.2 Pertahanan pada membran sel

Pada membrane sel terdapat peran vitamin E, beta karoten dan ko-enzim Q. Terbentuknya radikal alfa tokoferol akibat reaksi dapat diubah kembali menjadi alfa tokoferol dengan mengubah asam askorbat (vitamin C) menjadi dehidroaskorbat. Kemudian dehidroaskorbat diubah kembali menjadi asam askorbat oleh glutation dan sistein yang terdapat dalam sitosol (Makmun, 2002; Suryohudoyo, 2007).

Tokoferol + RO. → Tokoferol. + ROH (Lipid alkoxyl rad.)
Tokoferol + ROO. → Tokoferol. + ROOH (Lipid peroxyl rad.)
2 Tokoferol. + Askorbat → 2 Tokoferol + dehidroaskorbat (DHA)
Dehidroaskorbat (DHA) + 2GSH → Askorbat + GSSG

3.3.6.3 Pertahanan ekstrasel

Pertahanan ekstrasel terdiri :

a. Logam bebas Fe dan Cu diikat oleh protein khusus yaitu transferin, laktoferin dan seruloplasmin.

b. Molekul dengan berat molekul rendah juga bersifat antioksidan seperti bilirubin, vitamin C dan asam urat.
c. Glutation (GSH) dan superoksid dismutase (SOD) ekstrasel.
Glutation (GSH) merupakan antioksidan utama pada paru, tetapi kadarnya meningkat pada perokok dan penderita PPOK. Hal ini mungkin disebabkan adanya eksaserbasi akut.

3.3 Protease-antiprotease

Telah lama diketahui bahwa berbagai protease dapat memecahkan komponen jaringan ikat, seperti elastin pada parenkim paru dan menyebabkan terjadinya emfisema paru akibat ketidakseimbangan protease-antiprotease. Elastin merupakan target utama protease dan kerusakan elastin menyebabkan paru kehilangan elastisitasnya.

Kerusakan elastin ini tidak dapat diregenerasi dalam bentuk aktif. Adanya degradasi elastin dapat dilihat dari peningkatan ekskresi desmosin. Saat ini telah ditemukan lebih dari 20 jenis MMP yang dapat mendegradasi semua komponen matriks ekstrasel (MES) seperti elastin, kolagen, proteoglikan, laminin dan fibronektin.

Matriks metaloproteinase dihasilkan oleh netrofil, makrofag alveolar dan epitel saluran napas. Didapatkan peningkatan Human kolagenase (MMP-1) dan gelatinase B (MMP-9) pada cairan lavase bronkoalveolar pada penderita emfisema dibandingkan dengan kontrol. Aktivitas MMP juga meningkat pada perokok, akibat dari sitokin inflamasi, oksidan dan enzim lain seperti *netrofil elastase*. Matriks metaloproteinase berperan pada proses remodeling jaringan melalui degradasi matriks ekstrasel (MES). Proses remodeling ini terjadi pada berbagai kondisi yang fisiologis seperti embriogenesis, penyembuhan luka, penyakit inflamasi, invasi tumor dan angiogenesis (Barnes dkk, 2003).

Efek protease ini dapat dihambat oleh antiprotease endogen yang utama yaitu *alfa1-antitipsin* (AAT), *Secretory Leucoprotease Inhibitor* (SLPI) dan *Tissue Inhibitor of Matrix Metalloproteinase* (TIMP). Stres oksidatif menyebabkan kerusakan pada AAT dan SLPI dan mempercepat pemecahan serat elastin paru (Taggart dkk, 2000). Elastin merupakan target yang paling penting berbagai protease dan kerusakan elastin menyebabkan paru kehilangan elastisitas *(loss of elasticity).* Kerusakan elastin tidak dapat diregenerasi dalam bentuk aktif. Pada awalnya perhatian lebih banyak ditujukan pada neutrophil elastase sebagai penyebab PPOK, tetapi kemudian ditemukan banyak bukti yang menunjukkan peranan penting dari MMP dalam patogenesis PPOK (Vernooy dkk, 2004).

3.3.1 Matriks metaloproteinase

Matriks metaloproteinase (MMP) merupakan enzim proteolitik utama yang terlibat dalam degradasi matriks ekstrasel karena kemampuannya memecah semua protein yang ada didalam MES.

Matriks metaloproteinase mempunyai karateristik sebagai berikut (Gibb dkk, 1999) :
1. Disekresi dalam bentuk laten dan aktivasi dengan melepaskan gugus aminoterminal propeptida.
2. Mengandung ion Zn pada bagian aktifnya dan dapat dihambat oleh *Ethylene diamine tetraacetic acid* (EDTA)
3. Mempunyai aktivitas merusak MES.
4. Dapat dihambat oleh tissue inhibitory of metalloproteinase (TIMP)

Matriks metaloproteinase merupakan *zinc endopeptidase* yang berfungsi mendegradasi matriks ektrasel. Matriks metaloproteinase disebut juga matriksin karena dapat menghidrolisis matriks ekstrasel (MES). Matriks metaloproteinase merupakan protease ekstrasel yang mengatur pertumbuhan, fisiologi dan memegang peranan penting pada waktu perkembangan embrio, reproduksi, tissue remodeling, penyembuhan luka, angiogenesis dan penyakit seperti ateroma, artritis, kanker dan destruksi ulkus.

Salah satu kelompok dari MMP yaitu gelatinase disamping mengandung Zn juga mengandung kalsium *(zinc and calcium dependent endopeptidases).* Enzim ini disekresi sebagai *zymogen* (pro-MMP) dan dapat diaktivasi oleh berbagai proteinase, Hg-organik dan aktivitasnya dapat dihambat oleh tissue inhibitor of metalloproteinase (TIMP) yang spesifik (Vu dan Werb, 2000; Visse dkk, 2003)

Struktur MMP terdiri dari 3 domain :
1. *N terminal propeptide*
2. *Catalytic domain*
3. C terminal

Matriks metaloproteinase merupakan enzim *zinc-endopeptidase* yang menggunakan ion Zn yang bersifat elektrofilik untuk memecah bagian dari MES. Pada keadaan fisiologis, inhibitor endogen mengontrol aktivitas MMP, sedangkan pada keadaan patologis terjadi ekspresi MMP yang berlebihan atau kontrol yang tidak adekuat dari TIMP (Pocketti dkk, 2009).

Terminal propeptida terdiri dari 80 – 90 asam amino, mengandung sistein yang dapat berinteraksi dengan atom Zn pada domain katalitik melalui tiol rantai samping. Pemutusan gugus terminal propeptida oleh protease menyebabkan aktivasi zimogen yang merupakan bentuk laten MMP (Massova dkk, 1998).

Domain katalitik mengandung dua ion Zn dan paling sedikit satu ion Ca. Salah satu ion Zn berada ditempat aktif dan ikut dalam proses katalitik, sedangkan ion Zn yang lain dan ion Ca berada didalam domain katalitik. Ion Zn katalitik diperlukan untuk aktivitas proteolitik MMP.

Semua MMP mempunyai 3 residu histidin yang bekerja sama dengan ion Zn pada katalitik (Massova dkk, 1998). Aktivasi MMP terjadi dengan melepaskan propeptida pada terminal amino. Sistein pada propeptida berinteraksi dengan Zn pada bagian aktif MMP. Gangguan interaksi ini menyebabkan terjadinya mekanisme tombol sakelar *(switch mechanism)* yang menyebabkan aktivasi MMP. Matriks metalloproteinase juga dapat diaktivasi oleh *chaotrophic agent*, MMP dan protease lainnya. *Hemopexin like domain* pada MMP juga mirip dengan hemopeksin plasma berfungsi untuk mengikat substrat dan interaksi dengan TIMPs.

Aktivitas katalitik MMP diatur pada berbagai tingkatan yaitu transkripsi, sekresi, aktivasi dan inhibisi. Inhibisi oleh TIMPs melalui ikatan pada domain katalitik sehingga menyebabkan hambatan aktivitas enzimatik. Pada gelatinase, TIMPs melekat pada bentuk zymogen dari enzim tersebut (Vu dan Werb,2000). Terdapat beberapa MMP yang dapat merusak matriks ekstrasel seperti kolagenase dan *neutrophil elastase* (MMP-8). Berdasarkan substratnya, MMP dapat dibagi menjadi 6 kelompok sebagai berikut (Visse dkk, 2003)

3.3.1.1 Kolagenase

Yang termasuk kedalam kelompok ini adalah MMP-1, MMP-8, MMP-13 dan MMP-18. Kolagenase aktif merusak fibril dari kolagen, gelatin dan mempunyai aktivitas yang tinggi terhadap *denatured collagen* (Vu dan Werb, 2000). Enzim ini mempunyai kemampuan untuk memecahkan kolagen interstitial tipe I, II dan

III pada lokasi 3-4 dari N terminal. Kolagenase ini juga dapat menghancurkan matriks ekstrasel dan matriks non ekstrasel (Visse dkk, 2003). Stromelin mendegradasi komponen non-kolagen dari matriks ekstrasel (Vu dan Werb, 2000).

3.3.1.2 Kolagenase netrofil (MMP-8)

Protease ini lebih dikenal dengan kolagenase netrofil, diproduksi oleh kondrosit, sel endotel, fibroblast pada sinovial dan berbagai sel kanker. Enzim ini akan mendegradasi semua dari 3 rantai alfa kolagen I, II dan III serta berbagai substrat non-kolagen, berperan penting pada berbagai inflamasi dan progresivitas kanker (Tanaka dkk, 2007). Pada penelitian Vernooy dkk (2004) didapatkan peningkatan bermakna aktivitas MMP-8 pada perokok yang menderita PPOK dibandingkan dengan perokok yang tidak menderita PPOK ($p<0.05$). Penelitian pada penderita *Hospital Acquired Pneumonia* didapatkan peningkatan MMP-8 dan MMP-9 sebanyak 10 kali lipat pada cairan lavase bronkoalveolar penderita *Pneumonia Hospital Acquired* (Hartog, 2003)

3.3.1.3 Gelatinase

Yang termasuk kedalam kelompok ini adalah gelatinase A (MMP-2) dan gelatinase B (MMP-9). Keduanya dapat menghancurkan *denatured collagen* seperti kolagen tipe IV dan gelatin pada membrana basalis.

3.3.1.4 Gelatinase A (MMP-2)

Gelatinase A (MMP-2) berperan pada proses :

- *Remodeling* jaringan, pertumbuhan, penyembuhan luka dan invasi tumor
- Respons terhadap sitokin, faktor pertumbuhan dan mitogen
- Upregelasi pada penyakit tertentu seperti Non-systemic vasculitis neuropathy

Aktivitas MMP-2 ini meningkat bermakna pada kelompok PPOK dibandingkan dengan kontrol (Vernooy dkk, 2004). *Gelatinase* A (MMP-2) juga berperan pada agregasi platelet, regulasi tonus vaskuler dan disfungsi miokardial setelah iskemia-reperfusi.

3.3.1.5 Gelatinase B (MMP-9)

Gelatinase B (MMP-9) disimpan dalam granula tersier sel netrofil (Chakrabarti dkk, 2006).
Protease ini mempunyai sifat sebagai berikut :

- Diekspresikan secara luas
- Upregulasi pada *Polymyositis* dan *Non-systemic vasclitis neuropathy*
- Penyakit : *Idiopathic multicentic osteolysis*
- Diduga berperan pada pelepasan endometrium pada menstruasi
- Secara fungsional berhubungan dengan angiogenesis pembuluh darah

Gelatinase B (MMP-9) merusak kolagen tipe IV, gelatin dan elastin (Visse dkk, 2003). *Gelatinase* B juga dapat mendegradasi kolagen tipe V, X, XVII, *denatured collagen*, fibronektin dan mempunyai kemampuan elastolitik (Mantino dkk, 1997).

Makrofag alveolar merupakan sel pertahanan terbanyak yang terdapat pada paru, mensekresi enzim elastolitik MMP-2, MMP-9 dan MMP-12. *Gelatinase* B (MMP-9) merupakan enzim elastolitik utama yang diproduksi oleh makrofag, sel netrofil dan eosinofil. Makrofag alveolar mempunyai peranan yang sangat penting dalam patogenesis emfisema (Ito dkk, 2005). Enzim ini berperan pada penyakit paru yang ditandai kerusakan jaringan paru, perubahan struktur alveolar dan perbaikan jaringan paru yang abnormal (Mantino dkk, 1997; Barnes dkk, 2003).

Aktivitas *Gelatinase* B (MMP-9) juga meningkat bermakna pada penderita PPOK dibandingkan dengan kontrol (Vernooy dkk, 2004). Penelitian yang dilakukan Mercer dkk, 2005 didapatkan peningkatan aktivitas MMP-9 dari 10,5 menjadi 74 ug/mL pada PPOK dengan eksaserbasi. Disamping itu juga didapatkan aktivitas MMP-9 meningkat sebanyak 10 kali pada cairan lavase bronkoalveolar penderita dengan *Hospital Acquired Pneumonia* (Hartog dkk, 2003).

Gelatinase B juga berpengaruh pada migrasi dan penyebaran sel yang merupakan mekanisme yang penting pada proses perbaikan *(repair)* epitel pernapasan. Pada usia pertengahan *(middle age)* kadar glukosa berkorelasi terbalik dengan aktivitas MMP-9. Aktivitas MMP-9 juga berkorelasi terbalik dengan kadar kolesterol, aktivitas plasmin dan elastase. Aktivitas MMP-9 akan berkurang dengan betambahnya umur (Paczek dkk, 2008).

3.3.1.6 Makrofag elastase (MMP-12)

Makrofag elastase (MMP-12) pertama kali ditemukan oleh Werb dan Gordon pada tahun 1975 pada makrofag peritonel tikus. Aktivitas MMP-12 hampir tidak terdeteksi pada makrofag alveolar yang normal, tetapi tidak demikian pada perokok. MMP-12 juga dapat terdeteksi dengan imunohistokimia dan hibridisasi insitu makrofag alveolar pada penderita emfisema, tetapi tidak terdapat pada paru orang normal.

3.3.2 Penghambat aktivitas MMP

3.3.2.1. Tissue Inhibitory Matrix Metalloproteinase (TIMP)

Aktivitas MMP pada umumnya diatur melalui transkripsi gen, sintesis pro-MMP, aktivasi pro-MMP pasca translasi, dan interaksi dengan *tissue inhibitory matrix metalloproteinase* (TIMP). Tingginya aktivitas MMP-9 pada penelitian ini mungkin disebabkan subyek penelitian merupakan perokok aktif dimana radikal bebas dapat mengaktivasi pro-MMP dan mendegradasi TIMP. Peningkatan MMP-8 dan MMP-9 diduga akibat menurunnya aktivitas TIMP akibat produksi yang menurun dan mutasi gen. Asap rokok dapat menginaktivasi TIMP dan menyebabkan penurunan produksi TIMP oleh makrofag alveolar.

Makrofag alveolar pada perokok dengan emfisema memproduksi lebih sedikit TIMP dibandingkan perokok tanpa emfisema (Pons dkk, 2005). Kerentanan genetik dan polimofisme gen diduga dapat menjadi penyebab menurunnya aktivitas TIMP (Hirano dkk, 2001, Tzortzaki dan Siafakas, 2011).

3.3.2.2 Obat Penghambat Aktivitas MMP

Terdapat obat-obatan dapat menginaktivasi aktivitas MMP-8 dan MMP-9 dengan cara mengikat atom Zn pada bagian katalitik yaitu :

2.1. Marimastat, kinerja oabat ini buruk terhadap antineoplastik
2.2. Rebimastat
2.3. Prinomastat, obat ini mampu menghambat aktivitas MMP 2,3 9,13 dan 14 dan banyak digunakan untuk memblokade metastasis tumor dengan mencegah degradasi protein pada MES dan angiogenesis
2.4. Tanomastat

3.4 Faktor genetik

Faktor genetik diduga memegang peranan penting pada patogenesis emfisema seperti yang terlihat pada sekelompok famili penderita emfisema. Prevalensi emfisema pada usia muda berbeda pada tiap kelompok ras tertentu. Penderita defisiensi AAT (PiZZ) dengan kadar AAT kurang dari 10% nilai normal,

dapat terjadi emfisema dini yang disebabkan oleh asap rokok. Prevalensi emfisema yang disebabkan faktor genetik hanya 1% akibat defisiensi AAT dan varian genetik lainnya dengan kadar AAT yang rendah. Beberapa peneliti mendapatkan adanya hubungan antara timbulnya emfisema dengan polimorfisme gen, tetapi hal ini tidak disokong oleh peneliti lainnya. Penelitian pada populasi di Taiwan didapatkan bahwa risiko timbulnya emfisema adalah 10 kali lebih besar populasi dengan polimorfisme gen tumor nekrosis faktor alfa (TNF- α), tetapi tidak demikian pada populasi British (Barnes, 2000).

Faktor genetik yang mempengaruhi terjadinya emfisema adalah defisiensi *alfa1-antitripsin* (AAT) yaitu suatu protein pada serum yang diproduksi oleh hati. Pada keadaan normal, AAT akan menghambat kerja enzim netrofil elastase yang bersifat destruktif. Pada umumnya AAT terdapat dalam jumlah yang cukup pada paru, aliran darah dan berguna untuk melindungi paru terhadap enzim proteolitik akibat proses inflamasi yang disebabkan oleh rokok, toksin inhalan dan infeksi.

Kadar normal AAT adalah 150-350 mg/dl. Selain kelompok defisiensi AAT terdapat juga kelompok disfungsi AAT (kadar normal tapi tidak berfungsi baik) dan kelompok nol AAT (AAT tidak terdeteksi dalam serum). Penurunan kadar serum AAT kurang dari 35% dapat menimbulkan emfisema (Suyatna, 1995). Dilaporkan bahwa ada populasi dengan defisiensi AAT yang diturunkan secara genetik, dapat berkembang menjadi emfisema panlobular pada usia muda dibandingkan emfisema pada umumnya. Di Amerika Serikat, frekuensi penderita emfisema dengan defisiensi AAT ini hanya 1% dari seluruh penderita emfisema (Barnes dkk, 2003).

KESIMPULAN :

1. Oksidan atau radikal bebas menyebabkan kerusakan ekstrasel, dinding sel dan intrasel,
2. Selain Glutation (GSH) terdapat beberapa antioksidan yg sangat berperan menetralisir efek oksidan, seperti Vitamin A, Vitamin E dan Vitamin C
3. Terdapat penurunan kadar GSH pada perokok dengan emfisema
4. Peningkatan aktivitas MMP-8, dan MMP-9 yang bermakna pada pada perokok dengan emfisema dibandingkan perokok tanpa emfisema.
5. Perlu dipertimbangkan pemberian obat-obatan yang dapat menginaktivasi aktivitas MMP-8 dan MMP-9 seperti obat-obatan yang dapat mengikat ion Zn yang aktif pada bagian katalitik seperti marimastat, rebimastat, prinomastat dan tanomastat.

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MEDICAL ROLE IN DISASTER MANAGEMENT: FOCUS ON DISASTER MEDICINE

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ABSTRACT

Introduction

In recent years, Disasters are increasing in frequency and intensity. The impact of large-scale disasters on society is very large. An advance response plan is very useful for disaster mitigation and disaster risk management. This presentation will focus on disaster medicine and outline the medical roles in disaster risk management.

What is a disaster from medical perspective?

A "disaster" is defined by World Health Organization (WHO) as an occurrence where normal conditions of existence are disrupted and the level of suffering exceeds the capacity of the hazard-affected community to respond to it. A situation that needs assistance from outside the afflicted area 1). In addition to quantitative and/or qualitative shortage of resources, organizational and operational shortcomings are emphasized as an important component in the definition of a disaster by Utstein-Style Template for uniform data reporting of acute medical response in disasters 2). In United Kingdom, the Advanced Life Support Group (ALSG) of a charity dedicated to saving life by providing training develops a training course and text of disaster medical response named MIMMS (Major Incident Medical Management and Support). From an in-depth medical perspective, the MIMMS defines a major incident as an incident where the NUMBER, SEVERITY, or TYPE of LIVE casualties, or by its LOCATION, requires EXTRAORDINARY resource. In other words, it is defined as an incident that presents a serious threat to the health of the community, or an incident that disrupts the health service 3).

Medical roles in disaster management

What is the difference between routine-base emergency medicine and medicine at disaster/MCI?
First, characteristic of medical care at the time of disaster is much less supply than demand strongly interrupt to continue regional health care compared with the demand and supply balance of daily emergency medical care. Second, Sometimes the infra-structure is destroyed, supply will further decline. Third, most of a disaster will occur after-hours. Finally, it will not be completed within the boundaries of the plan of local government.

From these viewpoints, one of most important medical roles in disaster management is to establish and distribute “a systematic and all hazard approach” to all related person and organizations. Based on the sharing basic conceptions of the practical approach individually, a flamework of multi-organizational medical response plan at disaster should be prepared. Vital elements of the plan are prevention, mitigation by resilience, and support for restoration. The second role is research on risk assessment of a disaster. Finally, education is also essential medical role in disaster medical management.

Figure 1. Key elements of the medical response plan at a disaster

Practice of systematic and all hazard approach; CSCATTT

The MIMMS has developed a systematic and all hazard approach named "CSCATTT" (Table 1). The "CSCATTT" is a collection of the initials of the seven elements of this approach. First "C" means command and control. The "S" represents safety, and second "C" means communication. The "A" is assessment. The template of assessment of "METHANE" report is available (Table 2).

The "TTT" is so-called 3Ts of triage, treatment, and transport. The order of "CSCATTT" is basically important. The former part as "CSCA" are components of medical disaster management, and the latter part as "TTT" are components of medical support at a disaster. The "TTT" should be provided efficiently after establishment of a medical disaster management composed according to the "CSCA" conception.

Table 1. Vital elements of a systematic approach at a disaster: CSCATTT approach 3)

- Disaster medical management
  - ➢ Command and Control
  - ➢ Safety
  - ➢ Communication
  - ➢ Assessment
- Disaster medical support
  - ➢ Triage
  - ➢ Treatment
  - ➢ Transport

Table 2. Items to configure the METHANE report of an assessment of a disaster 3)

M	Major incident STANDBY or DECLARED
E	Exact location (grid reference)
T	Type of incident
H	Hazards, present and potential
A	Access, and egress
N	Number and severity of casualties
E	Emergency services, present and required

PHARMACOLOGICAL POINT OF VIEW OF IMMUNOTHERAPY FOR RECURRENT RTI IN EVACUATED DISASTER VICTIMS

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ABSTRACT

Introduction
Immunotherapy is a medical intervention aimed to prevent infection, or to shorten the length of illness, through strengthening the immune system. Disaster victims after evacuation have to stay for quite a long time in a non- permanent housing, usually the housing is crowded. In a crowded place the incidence of respiratory tract infection is high since it is a communicable disease that is airborne. For those with low immune status, the risk of recurrence is probably very high. The longer they stay the higher the risk, and recurrence will worsen their health condition. The worse the health condition the higher the risk for getting ill again and again; an intervention is needed to stop or decrease the risk of recurrence. Without prevention of recurrence, health condition will deteriorate, risk of complication is high, and with all the limitation of a disaster evacuation center morbidity, mortality and health cost will increased.

Recurrent Respiratory Tract Infection
Recurrent respiratory tract infection mainly involves the upper respiratory tract, and is associated with fever, cough, sore throat, and rhinitis; happens mostly in children. In certain condition it can also involving the lower respiratory tract, such as pneumonia. More than six serious diseases a year are defined as recurrent respiratory tract infections. Recurrent episodes should be separated by at least a two-week period with no symptoms.

Immunotherapy
Immune system is a complicated system, interaction of each component and factors that affecting it is not fully understand yet. How the Immune system really works is not fully understood up to now. The immune system is the first line of defence against an alien microorganism entering the body. The stronger the immune system, lesser would be the chance of falling ill.

On the whole, the immune system is defending against disease-causing microorganisms. But sometimes it fails: a germ invades successfully and makes sickness. Is it possible to intervene in this process and boost the immune system?

The idea of boosting immunity is inspiring, but the ability to do so has not been proven for several reasons. The immune system is a system, it requires balance and harmony to function well. Better understanding of how the many pathways involved act at once, and how is the interconnections of the immune response is needed.

If the concept of boosting immunity means increasing the numbers of immune cells, it is actually makes little sense scientifically. Boosting the number of cells, immune cells or others, is not necessarily a good thing for the body. Attempting to boost the cells of the immune system is especially complicated because there are so many different kinds of cells in the immune system that respond to so many different microbes in so many ways. Which cells should be increased, and to what number? No one knows the answer. What is known is that the body is continually generating immune cells. Certainly it produces many more

lymphocytes than it can possibly use. The cells remove themselves through apoptosis, some before they do any action, some after fighting the germs. No one knows how many cells, what best mix of cells, and exactly when, will make the immune system function at its best.

Pharmacological point of view

Pharmacological treatment can be preventive or curative, or symptomatic. In the case of recurrent infection it would be preventive. To prevent infection the best way is stay away from the germs, which is difficult in the situation of disaster victims evacuation. Evacuation centre most of the time is a crowded emergency housing; almost impossible to have a separated section for the sick person to prevent transmission. Infection cannot be prevented, so sickness should be prevented, most probably by boosting the immune system.
Is it possible to boost it with medicine ? Some medical advice are popular; such as keeping a good life style with well-balanced diet, exercise, no psychological stress etc. For now, there are no scientifically proven direct links between lifestyle and enhanced immune function. Study showed that lack of sleep can reduce the activity of T cells in the body. From sleeping for eight hours to walking in the sun to eating a balanced diet, the ways are simple but almost impossible to be followed by disaster victims.

Healthy immune system need good nourishment. Scientists have long recognized that people who live in poverty and are malnourished are more vulnerable to infectious diseases. Whether the increased rate of disease is caused by malnutrition's effect on the immune system, however, is not certain. There are still relatively few studies of the effects of nutrition on the immune system of humans, and even fewer studies that tie the effects of nutrition directly to the development of diseases.

A form of malnutrition that is surprisingly common even in affluent countries is "micronutrient malnutrition." Micronutrient malnutrition, in which a person is deficient in some essential vitamins and trace minerals is found common in the elderly and others who eat less and have less variety in their diets. People of all ages affected by disaster are probably, after some time, be affected by micronutrient malnutrition; especially if they were already eat less or have less variety in their diet in their past. There are some evidence that various micronutrient deficiencies, for example, deficiencies of zinc, selenium, iron, copper, folic acid, and vitamins A, D, C, and E , alter immune responses in animals. However, the impact of these immune system changes on the health of animals is less clear, and the effect of similar deficiencies on the human immune response has yet to be assessed.

If the diet is not providing all micronutrient needs, taking daily multivitamin and mineral supplement may bring other health benefits, beyond any possibly beneficial effects on the immune system. Using vitamin and mineral supplements provide the necessary nutrients.

However, studies showed that taking mega doses of a single vitamin did not give extra benefit, more is not necessarily better.

Many supplements and herbal products claim to boost or support immunity. In clinical pharmacology, scientific evidence in human is needed to support claims. Some of these claims are supported by some evidence, but not the best proof of cause effect relationship yet. Although some preparations have been found to alter some components of immune function, there is no evidence that they actually boost immunity to the point that protect against infection and disease. Demonstrating whether an herb, or any substance, can enhance immunity is a highly complicated matter. It is not known that , for example, whether an herb that seems to raise the levels of antibodies in the blood is actually doing anything beneficial for overall

immunity. While some changes in the leucocytes, lymphocytes or antibodies levels have been recorded, immunologists do not yet know what these changes mean in terms of human immune response. Results of immunotherapy to prevent RTI were studied mostly by evaluating the increase in cells involved in immune response. Measuring the potency to increase lymphocytes and immunoglobulins without any challenge is not enough, the experimental method should include a challenge test. By giving exposure to the agent of infection we can be sure that the response measured is due to the agent, not to the medicine.

The best evidence for cause-effect relationship is showed by experimental studies. But it is hard to perform "controlled experiments" in human beings. In a controlled experiment, only one factor is different between two groups, such as a certain herbs extract, and then measure the effect of that extract on some other measurable phenomenon, such as the amount of antibodies produced by a particular type of immune system cell as well as the ability of the whole human body to resist a certain infectious agent and prevent illness. In a human being, that kind of control is just not possible, since there are so many other things happening to the person during the time that the experiment was run. Besides, to see the effect to protect the body against a disease, the person has to be exposed to the agent/ cause of the disease. This is called “a challenge test” , to test the efficacy of the herbal against a certain infectious disease. A natural exposure or a natural challenge to the risk would be needed, since it is for sure unethical to expose someone to the risk purposely.

The clinical trial of Phylanthus niruri for haj pilgrims by Indonesian Ministry of Health showed that it was effective in decreasing the incidence of cold among the Indonesian haj pilgrims if given on top of regular multivitamin, compared to if given alone. This method using haj pilgrims satisfy the recommended method of exposure to challenge; it is ethical since the challenge is real and natural, haj pilgrimage exposes everyone to high risk of respiratory tract infections. Even though what cells are affected in the immune system was not studied, in clinical pharmacological point of view the evidence can be used to support the claim.

What is the best therapy for boosting immunity ?

Eating right is the best step to prevent illness. The old saying, “An apple a day can keep the doctor away,” may have truth behind it. Taking regularly foods rich in certain vitamins might help the immune system fight off illness. Which vitamins and minerals are actually related to immune systems ? There are vitamins that is believed to be the best for boosting immunity. But a strong scientific evidence is still lacking. There were studies to prove the claim, but most did not measure the vitamin status at the start of the study, or the effect were significant statistically but not clinically. Most of the studies showed that a good effect can be seen when vitamin treatment is given to those with vitamin insufficiency.

Lack of vitamin C make some people more prone to getting sick, especially infection. So does vitamin A; both vitamins are needed for a healthy mucous membrane of respiratory track and also the gastrointestinal. A study in Aceh, Indonesia, gave proof that giving vitamin A to school children in low socioeconomic community increase their immunities, showed by the significant decrease of RTI and diarrhoea incidence. It was detected beforehand that children in that area were lacking vitamin A. Since then, giving vitamin A to children at the Posyandu every 6 months has become a government program.

It’s also important to remember that a strong immune system is built by maintaining healthy eating habits over time. Eating four apples at breakfast or taking mega dose of supplement is not going to protect against catching RTI on that day.

Over the last 10 years, a number of studies have suggested that a dose higher than RDA of supplements are better in this case.

Vitamin C

A systematic review of vitamin C trials with military personnel and with other subjects living under conditions comparable to those of military were analysed to find out whether vitamin C supplementation can prevent respiratory infections. There were 12 trials in total, seven trials with military personnel, three trials with students in crowded lodgings, and two trials with marathon runners. Eight of these trials were double blind and placebo controlled and seven were randomized. Five small trials found a statistically significant at least 45 % reduction in the incidence of common cold in the vitamin C group. These trials were short and the participants were under heavy exertion during the trial. Another three trials found a statistically significant 80% reduction in the incidence of pneumonia in the vitamin C group. These positive findings are not strong because of the limitation value of the method, but can be used for consideration of the use of vitamin C in prevention of respiratory infections in a high risk population in a crowded setting like military barracks, in this case a disaster evacuation center.

Another report was from a meta-analysis of placebo controlled trials using at least 0.2 gr vitamin C; years 1966 up to 2012. Twenty-nine trial involving 11,306 participants contributed to the meta-analysis on the risk ratio (RR) of developing a cold whilst taking vitamin C regularly over the study period. In the general community trials involving 10,708 participants, the significant pooled RR was 0.97. Five trials involving a total of 598 marathon runners, skiers and soldiers on subarctic exercises yielded a significant pooled RR of 0.48 The majority of included trials were randomised, double-blind trials. The exclusion of trials that were either not randomised or not double-blind had no effect on the conclusions.

The failure of vitamin C supplementation to reduce the incidence of colds in the general population indicates that routine vitamin C supplementation is not justified, yet vitamin C may be useful for people exposed to brief periods of severe physical exercise such as marathon runners, skiers and soldiers.

Nevertheless, given the consistent effect of vitamin C on prevention of flu in certain types of communities, and the low cost and safety, it may be worthwhile for evacuated disaster victims to take at least 200 mg/day of vitamin C for the prevention of recurrent RTI, as well as for the other victims to prevent RTI. Further RCTs are warranted.

Vitamin D

In addition to its important role in skeletal development and maintenance, there is increasing evidence that vitamin D has a beneficial effect on other tissues. There are cells in the brain, heart, stomach, pancreas, lymphatics, skin, gonads, and prostrate, including T and B lymphocytes, that express the vitamin D receptor (VDR). In these tissues, vitamin D is thought to have roles in the improvement of immune function and the reduction of inflammation. The question of whether taking vitamin D supplements helps guard immunity has been controversial. Even though epidemiological studies found that vitamin D can help prevent diseases, it is still not convincing that there is a benefit of taking a supplement for people who are not deficient. There are studies with the objectives of finding the proof that vitamin D can help reduce the risk of respiratory infections, especially among people who do not get enough vitamin D from diet or exposure to sunlight. Staying out in the natural light for 10–15 minutes is one of the major contributors to the production of Vitamin D in the body. Low level of Vitamin D in the body has been termed as one of the major reasons for respiratory problems.

Accordingly, there is accumulating evidence that consumption of vitamin D may reduce respiratory tract infection (RTI) susceptibility in children. Initially, the available data support the link with tuberculosis (TB) risk, but there are now studies that support a connection with several others RTIs, such as recurrent acute otitis media (AOM), pharyngotonsillitis, rhinosinusitis, bronchiolitis and pneumonia.

Several recent studies have shown that vitamin D has different immunomodulatory properties associated with the risk of RTIs in children. In this regard, it is very important to understand the definition of deficiency of vitamin D. A level of at least 10 ng/mL 25-hydroxycholecalciferol (25[OH]D) is thought to be necessary to promote bone mineralization, and a concentration between 20 ng/mL and 50 ng/mL is considered adequate to provide an immunomodulatory effect; unfortunately there is no consensus yet.

In US, many food has long been fortified with vitamin D including milk and other dairy products. Institute of Medicine (IOM) guidelines were based on evidence for bone health. According to IOM recommendations, most adults need 600 IU vitamin D per day. Elderly starting from 70 years old are advised to increase the intake to 800 IUs per day. Based on safety profile, the IOM says that adults should not take more than 4,000 IU a day.

A systematic review and meta-analysis of randomised, double blind, placebo controlled trials were set out by an international team of researchers to assess the overall effect of vitamin D supplements on risk of acute respiratory tract infection.

Up to 2015 they collected data from 25 eligible trials (total 11 321 participants, aged 0 to 95 years). After adjusting for other potentially influential factors such as age, sex and study duration, vitamin D supplementation significantly reduced the risk of acute respiratory tract infection among all participants about 10% (adjusted odds ratio 0.88). Among those receiving daily or weekly vitamin D, protective effects were significantly stronger in those with baseline 25-hydroxyvitamin D levels <25 nmol/L, who had been vitamin D deficient when they enrolled in the studies (adjusted odds ratio 0.30). This result showed that people with very low vitamin D blood levels do better when they are given supplements . It is not surprising, cause if there is deficiency, getting an adequate amount will make a difference. The body of evidence contributing to these analyses was assessed as being of high quality. Vitamin D supplementation was safe and it protected against acute respiratory tract infection overall. Patients who were very vitamin D deficient experienced the most benefit. This type of research provides the strongest evidence for drawing causal conclusions because it draws together all of the best evidence.

In a linked editorial, two researchers said that a clinically useful effect remains uncertain and requires confirmation in well-designed adequately powered randomised controlled trials.

Maintenance of adequate vitamin D status may be an effective and inexpensive prophylactic method against some RTIs, but the supplementation regimen has not been clearly defined. Further clinical trials are needed to determine the blood vitamin D (25(OH)D) level associated with an increased risk of RTIs, and optimal vitamin D supplementation regimen according to the type of RTI, while also taking into consideration vitamin D receptor polymorphisms.

Vitamin A

Vitamin A deficiency is a public health problem in many parts of the world, particularly Africa and South-East Asia. Clinical trials have demonstrated that vitamin A supplementation reduces the severity of respiratory

infections in children with measles. However results from studies in children without measles suggest that vitamin A has protective effect only in children suffering from acute or chronic malnutrition.

A longitudinal prospective study of risk factors contributing to vitamin A deficiency and xerophthalmia revealed a dose-response relationship between the severity of mild pre-existing vitamin A deficiency and the subsequent incidence of respiratory infection (relative risk 2.0) , a prominent cause of child mortality. Subsequent community-based prophylaxis trials of varying design confirmed that vitamin A supplementation of deficient populations could reduce childhood (1-5 years old) mortality by an average of 35%. It is now estimated that improving the vitamin A status of all deficient children worldwide would prevent 1-3 million childhood deaths annually.

A literature review was done to assess the effectiveness and safety of vitamin A for preventing acute Low Respiratory Tract Infections in children up to seven years of age. Ten studies up to 2010 including 33,179 participants were included in this review. Eight studies found no significant effect of vitamin A on the incidence of acute LRTI. Two studies reported that vitamin A significantly reduced the incidence of acute LRTI in those with poor nutritional status, but increased the incidence in healthy children which is an unexpected result. Accordingly, vitamin A should not be given to all children to prevent acute LRTIs.

Despite its benefits in preventing diarrhea, vitamin A supplementation has only a limited effect in preventing acute LRTIs. Some evidence shows benefit are limited for children with low serum retinol or with a poor nutritional status. Limitations of this review include trials conducted within very specific populations and poor methodological quality of some of the included trials. Low dose vitamin A appears to have fewer side effects, and equal benefit to a high dose of vitamin A.

Herbal medicine

Herbal medicines are particularly regarded as an alternative or complement to conventional pharmaceuticals in the treatment and prevention of respiratory tract infections. Some herbs such as Andrographis, Echinacea, and Phyllantus were used traditionally to help prevent respiratory tract infections, as well as reducing the duration and severity of illness.

Randomized control trials suggest that Andrographis helps reduce symptoms of common cold, and may be as effective as paracetamol-based medicines. But there is no robust study to support its effectiveness in preventing cold.

The study on Echinacea so far fail to show its preventive effect for flu or common cold. Echinacea products vary widely, containing different species, parts, and preparations of the echinacea plant. Therefore it is hard to assess the efficacy because each study use different part of the plant. Echinacea has not been shown to reduce the number of colds that adults catch. Only a small amount of research on echinacea has been done in children, and the results of that research are inconsistent.

A meta-analysis study was run using data from randomized controlled trials up to 2015 that compared herbal therapy for RTI with no treatment, placebo, or any pharmaceutical medication in age 0 to 18 years. Eleven trials with 2181 participants were included. No clear evidence for Echinacea in preventing RTI symptoms was found. In one large clinical trial in children, those who took echinacea had an increased risk of developing rashes.

Conclusion:
Guidance are needed at evacuation center for workers and residents to help prevent transmission of contagious diseases by personal hygiene, environment hygiene, less stress, good food, good sleep, and enough drinking water.

As for immunotherapy, a blanket program of vitamins supplementation as therapeutic agent to fulfil the micronutrient basic need will further protect against infection because of a better immune system. Hippocrates said "Thy food is thy medicine"; experts say it's best to get vitamins through food rather than supplements. The situation in managing health of evacuated victims is an urgent situation. Moreover, in developing countries we might have to assume that every single person is in a micronutrient deficient condition. There is no time to catch up using food, the health condition should be treated, anything given should be as treatment. Vitamin and mineral (if any) should be considered as medicine. This is the time that food should be given as medicine. Another consideration in the case of crowded disaster emergency centre is the difficulties to ensure consistent availability of clean fruits and veggies, so it is more practical and safe to give supplements.

From pharmacological point of view at this point vitamin D has sufficient evidence of relation to immune response and relatively safe, it can be given in a blanket mode, in which case every single person should be treated. As for vitamin C, the consistent effect on prevention of flu can be seen only for certain communities with strenuous activity and living in a crowded place, it is not useful for general population. Since evacuation shelter and the living condition of disaster victims are having similarities with that characteristic, and the low cost of vitamin C and its safety, it may be worthwhile for evacuated disaster victims to take high dose vitamin C for the prevention of recurrent RTI, as well as for the other victims to prevent RTI. A blanket program of vitamin C 200 mg or more can be recommended. For the best evidence further randomised controlled trials are warranted in this condition.

For Vitamin A since there are report of negative effect, the use of low dose should be restricted for those who are most probably in a hypovitaminosis A condition, especially malnourished children. If in any case priority is needed due to shortage, then the ones with the worst nutrition condition should get the priority for vitamin D, vitamin C and A supplementation.

Available herbals with some probable evidence could be given if cost effective, further studies are needed for a stronger evidence. For now Phylanthus niruri has the best evidence among available herbals in Indonesia for a blanket program. Operational research for other indigenous herbals would be very useful as the subjects in the shelter are in a natural continuous exposure to high risk of recurrent RTI; which ethically may never be simulated in a normal population.

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Research on risk assessment

To fail to plan is to plan to fail. Planning guidance must include "all hazard". Familiarity with existing plans of ambulance service, hospital, special incident and other emergency service plans is very important and necessary for planning. Prior to the planning, familiarity with local high-risk sites is strongly required to make a hazard map.

Recently in Japan, a multilateral, multidisciplinary disaster management plan has been considered for future earthquakes with an epicenter in southern Tokyo. Then, we have developed a novel indicator of a degree of surge capacity during catastrophic event named "medical risk-resource-ratio (RRR)" for regional disaster medical response plan. We investigated the balance of medical risks and resources for each base disaster-response hospital in this area 4)5)6). Based on the government report outlining the postulated damage caused by the scenario earthquake, we calculated the medical RRR for each base disaster hospital in Yokohama. The RRR was 20.0 ± 10.4 per hospital bed and differed statistically among the hospitals. Differences in RRR can be used to clarify the relative burden of each base disaster hospital at the time of a postulated disaster. Further study is needed to quantify the resources that should be provided to each hospital and prioritize hospital support.

Education

In our country, open access education course for medical staff has been provided by the MIMMS-Japan. Since 2003, the courses not only of out-of-hospital response (the MIMMS course) but in-hospital response (the Hospital MIMMS course) have been frequently held, respectively.

In summary, from a viewpoint of "medicine at the time of disaster" rather than "disaster medicine", we should objectively observe the status and grasp its various aspects. The "CSCATTT" approach will greatly contribute to the practice of medical response at the time of disaster. Risk assessment and education are nuts and bolts to prepare the response plan. Based on the "CSCATTT" approach, daily routine and disaster emergency medical response are essentially same to practice a medical management and support.

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RESPIRATORY CARE ON VOLCANO ERUPTION AND FIRE

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ABSTRACT

Introduction

Indonesia is one of the countires with the most active volcanoes in the world . Vulcanoes are part of a series of active fire mountains known as the ring of fire. In the recorded history of vulcanoes in the world, two major eruptions were recorded in Indonesia namely the eruptions of Mount Tambora and Mount Krakatau. The eruption of Mount Tambora in 1815 resulted in the spread of vulcanic ash throughout the earth. Another impact, in 1816 it was known as the year without summer in the northern hemisphire. Mount Krakatau wich erupted in August 1883, had a well known impact throghout the world. The dust clouds crossed the world several times and incident triggered a tsunami capable pushing warship hundreds of meterto land. Meanwhile, one of the most erupting vulcanoes in Indonesia is Mount Merapi. This mountain has been active since 1900 until now with short periods of rest or rest (on average no more than 3,5 years). Mount Merapi is known to have an eruption cycle for 3.5 years, but the cycle is only statistical calculation. So, more than 100 times the eruption of Mount Merapi can occur within 1 to 18 years. That means the eruption of Mount Merapi in one or two years can also occur. In short, the eruption of Mount Merapi is a permanent disaster threat. Indonesia's position at the confluence of mainplates, namely the Eurasian, Indo-Australianand Pasific plates. Thus, as a country located in the world fire ringlane, Indonesia has many volcanoes, both active and volcanic wich have not shown volcanic activity for a long time. Indonesia has around 129 active volcanoes, wich is about 179%of all volcanoes active throught the world.[6]

Indonesia lies between the Ring of Fire along the northeastern islands adjacent to and including New Guinea and the Alpide belt along the south and west from Sumatra, Java, Bali, Flores, and Timor. The famous and very active San Andreas Fault zone of California is a transform fault which offsets a portion of the East Pacific Rise under southwestern United States and Mexico. The motion of the fault generates numerous small earthquakes, at multiple times a day, most of which are too small to be felt.[10]

Volcanoes and their eruptions can result in a wide range of health impacts, arguably more varied than in any other kind of natural disaster. At least 500 million people worldwide live within potential exposure range of a volcano that has been active within recorded history. Volcanic activity can also affect areas hundreds or thousands of kilometres away, as a result of airborne dispersion of gases and ash, or even on ahemispheric to global scale due to impacts on climate.[1]

Eruption

Eruption that emit volcanic material in the form of gas, dust, lava flows, rock, fragments and others, eruptions can be classified based on where the material comes out.

1. Central eruption, eruption out through the main crater
2. Side eruption, reruption out through out of the slope of the vulcano
3. Slit eruption, eruption coming out of long cracs can reach up to how many kilometers
4. Extrensic eruption, the eruption that comes out from the side of the magma kitchen through the crater itself. [7]

Volcanic phenomen and health hazard

Volcanic ash consists of fragments of pulverized rock, minerals and volcanic glass, created during volcanic eruptions and measuring less than 2 mm (0.079 inches) in diameter. The term volcanic ash is also often loosely used to refer to all explosive eruption products (correctly referred to as tephra), including particles larger than 2mm. Volcanic ash is formed during explosive volcanic eruptions when dissolved gases in magma expand and escape violently into the atmosphere. The force of the escaping gas shatters the magma and propels it into the atmosphere where it solidifies into fragments of volcanic rock and glass. Ash is also produced when magma comes into contact with water during phreatomagmatic eruptions, causing the water to explosively fl ash to steam leading to shattering of magma. Once in the air, ash is transported by wind up to thousands of kilometers away. Due to its wide dispersal, ash can have a number of impacts on society, including human and animal health, disruption to aviation, disruption to critical infrastructure (e.g., electric power supply systems, telecommunications, water and waste-water networks, transportation), primary industries (e.g.,agriculture), buildings and structures.[2]

Formation vulcanic ash

Volcanic ash is formed during explosive volcanic eruptions, phreatomagmatic eruptions and during transport in pyroclastic density currents. Explosive eruptions occur when magma decompresses as it rises, allowing dissolved volatiles (dominantly water and carbon dioxide) to exsolve into gas bubbles. As more bubbles nucleate a foam is produced, which decreases the density of the magma, accelerating it up the conduit. Fragmentation occurs when bubbles occupy ~70-80 vol% of the erupting mixture. When fragmentation occurs, violently expanding bubbles tear the magma apart into fragments which are ejected into the atmosphere where they solidify into ash particles. Fragmentation is a very efficient process of ash formation and is capable of generating very fine ash even without the addition of water. Volcanic ash is also produced during phreatomagmatic eruptions. During these eruptions fragmentation occurs when magma comes into contact with bodies of water (such as the sea, lakes and marshes) groundwater, snow or ice. As the magma, which is significantly hotter than the boiling point of water, comes into contact with water an insulating vapor film forms (Leidenfrost effect). Eventually this vapor film will collapse leading to direct coupling of the cold water and hot magma. This increases the heat transfer which leads to the rapid expansion of water and fragmentation of the magma into small particles which are subsequently ejected from the volcanic vent.Fragmentation causes an increase in contact area between magma and water creating a feedback mechanism,leading to further fragmentation and production of fine ash particles. Pyroclastic density currents can also produce ash particles. These are typically produced by lava dome collapse or collapse of the eruptioncolumn. Within pyroclastic density currents particle abrasion occurs as particles interact with each other resulting in a reduction in grain size and production of fine grained ash particles. In addition, ash can be produced during secondary fragmentation of pumice fragments, due to the conservation of heat within the flow. These processes produce large quantities of very fine grained ash which is removed from pyroclastic density currents in co-ignimbrite ash plumes.[2]

Characteristics of ash particles

Chemical

The types of minerals present in volcanic ash are dependent on the chemistry of the magma from which it erupted. Considering that the most abundant elements found in magma are silica (SiO_2) and oxygen, the various types of magma (and therefore ash) produced during volcanic eruptions are most commonly explained in terms of their silica content. Low energy eruptions of basalt produce a characteristically dark coloured ash containing ~45 - 55% silica that is generally rich in iron (Fe) and magnesium (Mg). The most

explosiv rhyolite eruptions produce a felsic ash that is high in silica (>69%) while other types of ash with an intermediate composition (e.g., andesite or dacite) have a silica content between 55-69%.

The principal gases released during volcanic activity are water, carbon dioxide, sulfur dioxide, hydrogen, hydrogen sulfide, carbon monoxide and hydrogen chloride. These sulfur and halogen gases and metals are removed from the atmosphere by processes of chemical reaction, dry and wet deposition, and by adsorption onto the surface of volcanic ash. It has long been recognised that a range of sulfate and halide (primarily chloride and fluoride) compounds are readily mobilised from fresh volcanic ash. It is considered most likely that these salts are formed as a consequence of rapid acid dissolution of ash particles within eruption plumes, which is thought to supply the cations involved in the deposition of sulfate and halide salts.[3,5]

Components

Volcanic ash particles erupted during magmatic eruptions are made up of various fractions of vitric (glassy, non-crystalline), crystalline or lithic (non-magmatic) particles. Ash produced during low viscosity magmatic eruptions. produce a range of different pyroclasts dependent on the eruptive process.

Morphology

The morphology of ash from eruptions of high-viscosity magmas (e.g.,rhyolite, dacite, and some andesites) is mostly dependent on the shape of vesicles in the rising magma before disintegration. Vesicles are formed by the expansion of magmatic gas before the magma has solidified. Ash particles can have varying degrees of vesicularity andvesicular particles can have extremely high surface area to volume ratios.

Grain size

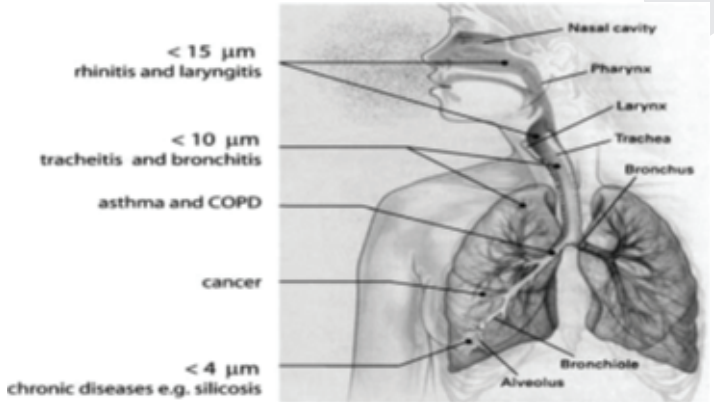
Volcanic ash consists of particles (pyroclasts) with diameters <2 mm (particles >2 mm are classified as lapilli), and can be as fine as 1 μm. The overall grain size distribution of ash can vary greatly with different magma compositions. The proportions of fine ash are higher for silicic explosive eruptions, probably because vesicle size in the pre-eruptive magma is smaller than those in mafic magmas.[4]

Impact in health hazard.

Tephra is a general term for any fragmentary material originally emitted from volcanoes, while ash refers to tephra particle, 2 mm across. Tephra presents several kinds of health hazard: through inhalation and abrasion of skin and conjunctiva, through building collapse from loading on roofs, and through impacts on terrestrial and aquatic environments. Volcanic ash is defined as pyroclasts 2 mm or less in diameter. The different physicochemical properties of ash are critical to its biological reactivity and hence its respiratory health effects, but relatively little is understood about the disease mechanisms at the cellular level. Freshly erupted ash differs from other natural dusts in several ways. The particle surfaces are un-weathered and are, therefore, not oxidised or leached and can carry condensed volatiles such as acids, polycyclic hydrocarbons and trace metals. Fine ash particles tend to fall in roughly spherical clusters (<100 μm), which readily break up on impact in dry conditions or when resuspended by vehicles and other human activity. Sulphuric and other acids adsorbed from the gases in the plume may be present on the surface of ash particles, potentially adding to the irritancy of the ash on the airways. The strong acids may react with the glass and silicate components of the ash particles, altering their surface characteristics and forming calcium sulphate and sodium chloride as precipitated coatings on the ash. The acid salts can also combine with rain in forming crusts on top of deposits which makes the ash less easily resuspended by wind. Sulphur dioxide (SO_2) or acid aerosols can trigger asthma attacks at very low concentrations in asthma patients so

grounding of the gas plume as a cause of respiratory symptoms always needs to be excluded. The grain size of ash particles is of critical importance and is conventionally defined in terms of the aerodynamic diameter. Particulate matter less than 10 μm diameter (PM10) is classed as thoracic, and respirable if less than 4 μm. The finer respirable particles can be breathed into the alveolar region of the lung and have the greatest toxic potential . Recent research has shown that fine particles (<1 μm), and ultrafines, (<0.01 μm), are likely to be the most toxic (Expert Panel on Air Quality Standards 1995), but whether this applies to volcanic ash is not yet clear. Until recently, volcanologists did not routinely analyse PM10 or PM4, thereby compounding the lack of information available for evaluating the health effects of eruptions. The reactivity of particles within the lung is related to the surface area and number of particles more than the mass of particles. It is, therefore, useful to quantify mineral assemblages in terms of number or surface area percent as well as weight percent . At the scale of the thoracic and respirable fractions, ash particles from eruptions of very different magma composition (e.g. basaltic and andesitic) are morphologically similar and it is not possible to determine composition of particles simply by observing the morphology.[5,8]

Fig.1 the airways.



the following factors emerged as potentially important predictors of the scale and nature of respiratory effects:

- Concentration and size of the ash particles inhaled, particularly the percentage of finer particles (here,4 mm and ,2.5 mm) able to penetrate deeply into the lung, and coarser particles (of 4–10 mm) chiefly impacting on the upper airways.
- Mineralogic composition, particularly the free silica content.
- Surface properties, especially Fe2+ content—higher iron resulting in more free radical generation in toxicological studies, with fresh ash generating more radicals than weathered samples.

In some eruptions, ash particles can be so fine that they are breathed deep into the lungs. With high exposure, even healthy individuals will experience chest discomfort with increased coughing and irritation. Common acute (short-term) symptoms include:

- Nasal irritation and discharge (runny nose).
- Throat irritation and sore throat, sometimes accompanied by dry coughing.
- People with pre-existing chest complaints may develop severe bronchitic symptoms which last some days beyond exposure to ash (for example, hacking cough, production of sputum, wheezing, or shortness

of breath).
- Airway irritation for people with asthma or bronchitis; common complaints of people with asthma include shortness of breath, wheezing and coughing.
- Breathing becomes uncomfortable.[8]

In rare circumstances, long-term exposure to fine volcanic ash may lead to serious lung diseases. For these diseases to occur, the ash must be very fine, contain crystalline silica (for the disease silicosis to occur) and the people must be exposed to the ash in high concentrations over many years. The fine ash particles irritate the airways and cause them to contract, making breathing more difficult in people who already have lung problems. The fine dust also causes the lining of the airways to produce more secretions which can cause people to cough and breathe more heavily. Asthma sufferers, especially children who may beheavily exposed to the ash when they play, may suffer bouts of coughing, tightness of the chest and wheezing. Some people who have never knowingly had asthma before, may experience asthma symptoms following an ashfall, especially if they go outdoors in the ash and over-exert themselves. The development of respiratory symptoms from the inhalation of volcanic ash depends on a number of factors. These include the concentration of particles in the air, the proportion of fine particles in the ash, the frequency and duration of exposure, the presence of crystalline silica and volcanic gases or aerosols mixed with the ash, and meteorological conditions. Existing health conditions and the use of respiratory protective equipment will also influence the symptoms experienced.

Chronic health effects from volcanic ash fall are possible, as exposure to free crystalline silica is known to cause silicosis. Minerals associated with this include quartz, cristobalite and tridymite, which may all be present in volcanic ash. These minerals are described as 'free' silica as the SiO2 is not attached to another element to create a new mineral. However, magmas containing less than 58% SiO2 are thought to be unlikely to contain crystalline silica. The main causes of death are heat induced fulminant shock, asphyxia due to plugs of ash in the airways, thermal lung injury, and deep thickness burns.5

Protect against ash and intervention to injury
- Seek the local wind and weather pattern.
- Avoid strenuous outdoor activities in areas with increased vog conditions.
- Close windows and doors, and remain indoors or upwind of the vog source.
- Use air conditioning and HEPA filters. In the same way that air conditioning removes water vapor, it will remove sulfate particles. A HEPA (PM2.5) filter provides additional protection.
- Shelter in pollution-free areas.
- Bronchoscopy was the potential therapeutic for bronchial toilets
- FOB was performed on initial assessment in a suspicion of inhalation trauma.

helps establish the severity of the injury and diagnose the inhalation injury.[4,9]

Summary
- Large eruptions can cause global climate change through entrainment of gas and particles into the upper atmosphere.
- Exposure to volcanic ash may result in acute respiratory morbidity, especially in those with pre-existing respiratory disease.
- Respirable particulates (<10mm in diameter—PM10) have been associated with increased respiratory symptoms respiratory-related emergency department attendances or hospital admission of asthma and COPD

- Eruptions of ash and discharges of aerosols and acid gases may be transported long distances (hundreds of kilometres) and cause health effects remote from thevolcano concerned.
- Major mortality associated with volcanogenic phenomena in recorded history has resulted from pyroclastic density currents

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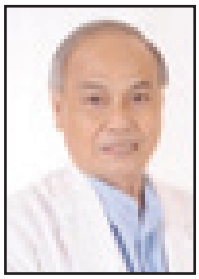
RESPIRATORY CARE ON VOLCANO ERUPTION AND FIRE



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ABSTRACT

The vulnerability of the Asia-Pacific and developing countries are multifactorial and ever a continuing present and continuing danger to the communities of the region. Various types of respiratory system injuries can occur at the time of a disaster.

In the event of natural disasters, such as earthquakes or volcanic eruptions, both traumatic and inhalation injuries may occur when a building collapses or catches fire. Tropical storms, floods, landslides, and tornadoes often cause traumatic lung injuries, as does drowning or near-drowning.

Man-made disasters, such as those involving transport and use of weapons of mass destruction, also cause inhalation and traumatic injuries, including blast lung injuries. There may be many patients with burns and inhalation injuries at the scene of a fire-related disaster. During the last three decades, the prognosis of patients with major thermal injury has improved significantly with advances in the overall care of burns patients. However, inhalation injury significantly increases mortality. Half of all fire-related deaths are attributable to smoke inhalation.

Further, there are a number of impediments to efficient care in disaster settings, including delayed rescue and transport services, poor access to medical supplies, poor initial assessment by physicians inexperienced in treating burns patients, and a shortage of medical resources. Even a patient with major burns may not have a high degree of urgency, but a patient with inhalation injury can deteriorate rapidly because of airway compromise and can be a true emergency.

Further, multisystem injuries occur in combination with those sustained by the respiratory system. Poor hygiene, air and water pollution, malnutrition as a result of food shortage, and limited medical resources in a disaster setting can aggravate the course of disaster- related injuries. Huge numbers of casualties occur at the scene of a disaster.

The principle in disaster medicine is to provide the best treatment to the greatest number of victims using limited medical resource. Victims with severe injuries or complications who need a large amount of medical resources should be transported to facilities outside the disaster zone for further treatment after vital signs are stabilized.

To ensure an integrated and effective response to disasters in the future, physicians should understand the fundamental principles of disaster medicine and participate in the disaster planning process.

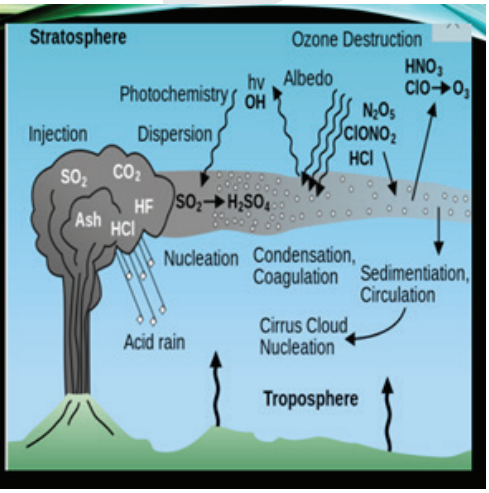
Respiratory Tract Burns, Traumatic Pulmonary Contusions, Crushing Death, and Crush Syndrome: What Kinds of Lung Injury Occur by Respiratory Tract Burn, Traumatic Contusion, and Crush Syndrome? *Hiroshi Imamura*

- Various types of respiratory system injury can occur in a disaster setting, including traumatic thoracic injury, traumatic asphyxia, and inhalation injury.
- Further, multi-system injuries may occur in combination with those affecting the respiratory system. Acute respiratory distress syndrome often complicates disaster-related injuries, such as crush syndrome, thoracic trauma, hemorrhagic shock, and burns.
- Poor hygiene, air and water pollution, malnutrition arising from food shortages, ,and shortages of medical resources in a disaster setting can aggravate the course of a disaster-related injury.
- A large number of casualties may occur at the scene of a disaster.
- The principle in disaster medicine is to provide the best treatment to the greatest number of victims using limited medical resources.
- Treatment of patients with severe injuries or complicated needs consumes a considerable amount of these resources. Therefore, victims should be transported to facilities outside the disaster zone as soon as possible for further treatment after vital signs are stabilized.

HAZARDS OF VOLCANIC ASH

A multitude of dangerous particals and gases, such as aerosols, are carried in volcanic ash. Some of these include;

- Carbon dioxide
- Sulfates (sulfur dioxide)
- Hydrochloric acid
- Hydroflouric acid

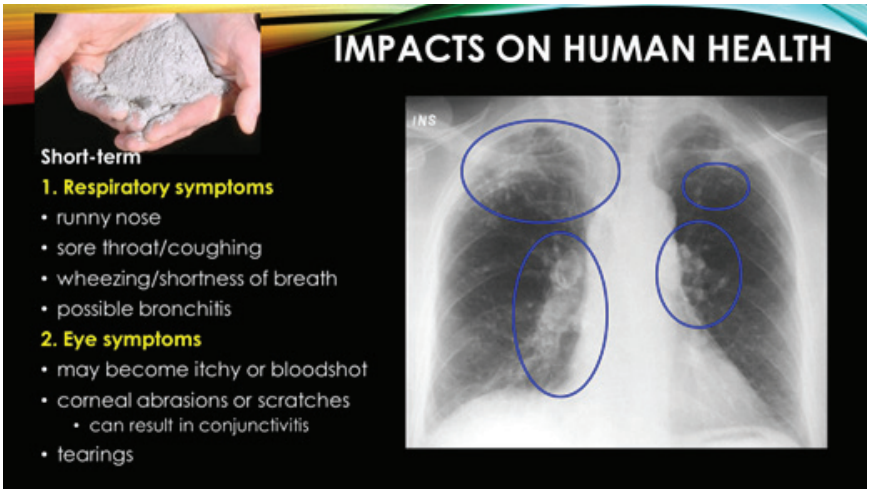


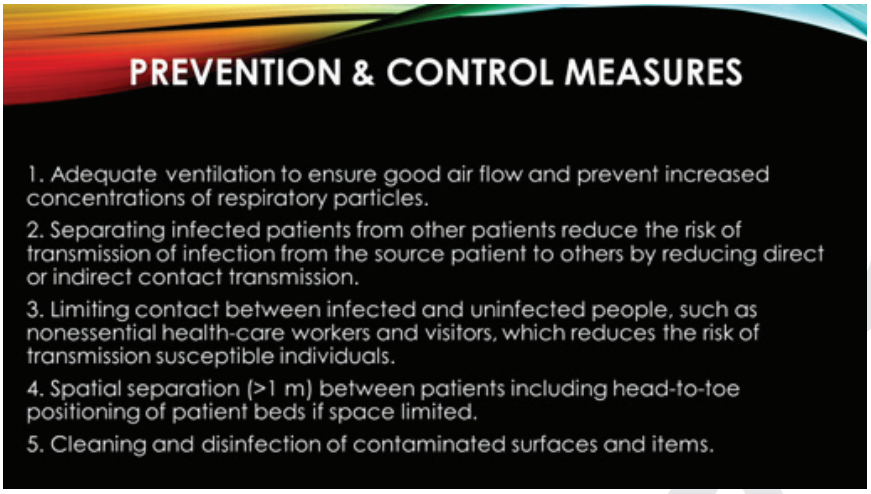
RESPIRATORY HEALTH ISSUES IN THE ASIA–PACIFIC REGION-NATURAL DISASTERS AND THE LUNG

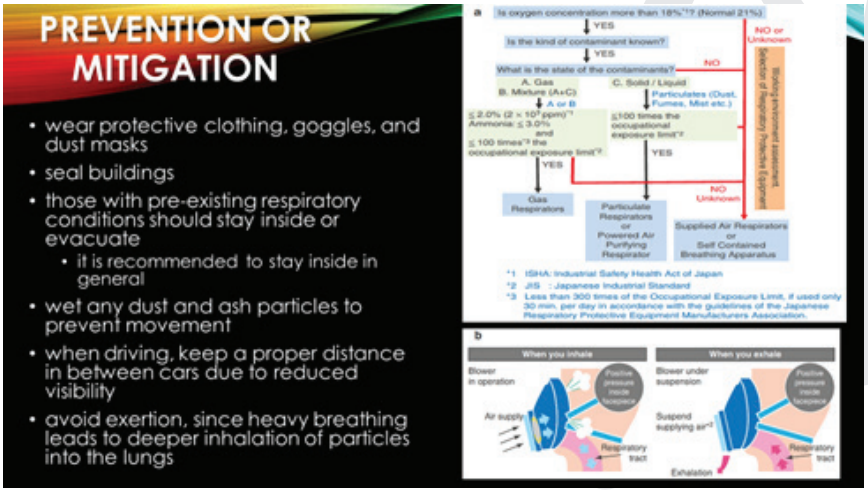
RESPIROLOGY_2011 BRUCE ROBINSON, MOHAMMAD FAHMI ALATAS, ANDREW ROBERTSON, HENRY STEER

- Inhalation of respirable particles, smoke or other
- Toxic gases
- Aspiration of water and water borne pathogens
- Direct trauma to the chest
- Psychological effects causing respiratory symptoms

- **DIRECT**
  - Inhalation injury / Acute lung injury
    - Haze / Smog / Volcanic emissions
  - Near-drowning / Aspiration syndrome
  - Dust / Building collapse
  - Traumatic chest injuries / Polytrauma / Crush
  - Acute anxiety – PTSD – Depression
- **INDIRECT**
  - Communicable resp-infections/ prevention
  - Collapse of health systems
  - Disaster preparedness & mitigation
  - Patient transport / Surveillance


IMPACTS ON HUMAN HEALTH
Short-term
1. Respiratory symptoms
• runny nose
• sore throat/coughing
• wheezing/shortness of breath
• possible bronchitis
2. Eye symptoms
• may become itchy or bloodshot
• corneal abrasions or scratches
• can result in conjunctivitis
• tearings


PREVENTION & CONTROL MEASURES
1. Adequate ventilation to ensure good air flow and prevent increased concentrations of respiratory particles.
2. Separating infected patients from other patients reduce the risk of transmission of infection from the source patient to others by reducing direct or indirect contact transmission.
3. Limiting contact between infected and uninfected people, such as nonessential health-care workers and visitors, which reduces the risk of transmission susceptible individuals.
4. Spatial separation (>1 m) between patients including head-to-toe positioning of patient beds if space limited.
5. Cleaning and disinfection of contaminated surfaces and items.


PREVENTION OR MITIGATION
• wear protective clothing, goggles, and dust masks
• seal buildings
• those with pre-existing respiratory conditions should stay inside or evacuate
• it is recommended to stay inside in general
• wet any dust and ash particles to prevent movement
• when driving, keep a proper distance in between cars due to reduced visibility
• avoid exertion, since heavy breathing leads to deeper inhalation of particles into the lungs
Is oxygen concentration more than 18%*1? (Normal 21%)
YES
NO or Unknown
Is the kind of contaminant known?
YES
NO
What is the state of the contaminants?
A. Gas
B. Mixture (A+C)
C. Solid / Liquid
Particulates (Dust, Fumes, Mist etc.)
A or B
Selection of Respiratory Protective Equipment
Work environment assessment
Gas Respirators
Particulate Respirators or Powered Air Purifying Respirator
Supplied Air Respirators or Self Contained Breathing Apparatus
*1 ISHA: Industrial Safety Health Act of Japan
*2 JIS : Japanese Industrial Standard
*3 Less than 300 times of the Occupational Exposure Limit, if used only 30 min. per day in accordance with the guidelines of the Japanese Respiratory Protective Equipment Manufacturers Association.
When you inhale
When you exhale
Blower in operation
Blower under suspension
Positive pressure inside facepiece
Air supply
Suspend supplying air*2
Respiratory tract
Exhalation

RESPIRATORY CARE ON TSUNAMI & FLOOD

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Department of Disaster Medical Management

ABSTRACT

Introduction

In 2018, there were 315 natural-disaster events, with 11,804 deaths and over 68 million people affected worldwide.1) Asia is particularly susceptible to natural disasters because of its geology in relation to earthquakes and high population density. Among various natural disasters, tsunami and floods have affected people the most, especially in Indonesia and Japan. Some recent examples of damaging events of tsunami and floods include the 2004 Indian Ocean (Sumatra) earthquake and 2011 Great East Japan Earthquake. The tsunami-hit area extended 1500 km from Indonesia to the Bay of Bengal in the 2004 Sumatra earthquake and more than 500 km along the northern east coast of Japan in 2011.2)

In the 2011 Great East Japan Earthquake, the deaths and missing tolls reached approximately 20,000, 92.5 % of which drowned. Many victims died from choking on seawater or sludge containing various objects (e.g., soil, sand, buildings, oil, etc.). An autopsy report stated that the drowning was not only because of seawater but also because of tsunami sands, and some referred to it as drowning by sands.[2] Most individuals who failed to escape from the approaching tsunami lost their lives, and even those who could be rescued developed serious systematic disorders called "tsunami lung" due to drowning.[3]

Definition of "tsunami lung"

The term "tsunami lung" was used for the first time after the 2004 Sumatra earthquake and tsunami disaster. Allworth[4] reported that patients with "tsunami lung" had persistent symptoms despite broad-spectrum antibiotic therapy and developed radiologic and clinical manifestations of necrosis with pleural involvement weeks after immersion in the tsunami. Potera[5] reported that "tsunami lung" occurs when people who are swept by tsunami waves inhale salt water contaminated with mud and bacteria, which results in pneumonia-like infections, and are treated with antibiotics. Both of them reported "tsunami lung" as an intractable bacterial pneumonia resulting in necrotizing pneumonia or pulmonary abscesses due to the *Burkholderia pseudomallei* in the Asian soil and seawater. However, after the 2011 Great East Japan Earthquake, the term "tsunami lung" came to be used in a broad sense. In this presentation, "tsunami lung" is collectively used for patients with the lung lesion and respiratory symptoms because of tsunami.

Pathophysiology and features of "tsunami lung"

Aspiration of water into the lung can lead to infection, loss of the alveolar surfactant, pulmonary edema, and acute respiratory distress syndrome (ARDS).[6] In addition, vomiting of the swallowed water can lead to aspiration of gastric contents, especially if consciousness and airway protective reflexes are impaired. Respiratory infection from near-drowning reflects the microbial flora of the aspirated seawater and colonizers of the oropharynx. It is necessary to consider the pathogen inhabiting the area for treating "tsunami lung". Table 1 shows the pathogens of respiratory infections associated with near-drowning.[3-5,7,8]

Table 1. Pathogens of infectious lung disease associated with near-drowning 3-5,7,8)

Gram-negative aerobic bacteria
- *Aeromonas*
- *Burkholderia pseudomallei*
- *Chromobacterium violaceum*
- *Francisella philomiragia*
- *Klebsiella pneumoniae*
- *Legionella*
- *Neisseria mucosa*
- *Pseudomonas aeruginosa*
- *Shewanella putrefaciens*
- *Vibrio*

Gram-positive aerobic bacteria
- *Streptococcus pneumoniae*
- *Staphylococcus aureus*

Fungi
- *Aspergillus*
- *Pseudallescheria boydii*

Moreover, many victims described the tsunami wave as being "black" in the 2011 earthquake. When intubation and suction of the near-drowning victims was performed, medical staff found sludge or slime in the extracted liquid. It means that drowning in a tsunami may differ markedly from drowning in normal seawater. Tsunami waves contained various substances, such as soil, sand, buildings, oil etc. These oils float on the sea surface because of the specific gravity of seawater. When people caught in a tsunami attempt to ascend to the sea surface to breathe, they are at a high risk of aspirating the oil. The lung invaded by oil develops serious chemical-induced pneumonia. In addition, as the aspirated foreign bodys (i.e. sand or mud) cannot be removed naturally and remains in the body for 1–2 weeks after the injury, respiratory symptoms caused by foreign bodies can easily become chronic.9)

As a result, "tsunami lungs" caused by aspiration of seawater and various substances including pathogens, oil, and mud may represent a combination of respiratory infections and chemical-induced pneumonia.[5)]

Respiratory care of tsunami lung

"Tsunami lung" is easily diagnosed because many patients have a drowning episode, respiratory symptoms, and residual abnormal shadow on chest imaging. If possible, it is desirable for examination of microorganisms to be carried out before starting an antimicrobial treatment. However, clinical diagnosis based on the medical history and clinical symptoms is preferred because blood examination and imaging are limited in a disaster setting. Antibiotics should be administered in patients who are clinically diagnosed with "tsunami lung", as a conservative approach is warranted because of the high morbidity associated with this complication and the likelihood of aspiration of contaminated seawater in most disaster settings.[10)] In general, "tsunami lung" that follows near-drowning results from a polymicrobial infection. Therefore, carbapenem is a first-line medicine in empirical antimicrobial treatment.[4)] Fungal infection can also complicate "tsunami lung" and should be considered in patients not responding to antibacterial therapy if they develop not only pneumonia but also brain abscess or meningitis. Voriconazole is a first-line medicine in empirical antifungal treatment.[8)] In addition, depending on the respiratory status of patients with "tsunami lung", oxygenation and mechanical ventilation therapy is carried out. As mentioned above, because the

aspirated foreign body (i.e. sand or mud) cannot be removed naturally, it is better to perform postural respiratory drainage and remove the foreign body from the lung using bronchoscopy.

Several months after tsunami, some patients have dyspnea on exertion, prolonged pneumonic shadows and allergic lung inflammation on chest imaging, such as hypersensitivity pneumonia or organizing pneumonia. This type of pneumonia may be observed in heavy equipment clean-up workers and recovery support volunteer workers because of the inhalation of wreckage dust or fungus. Sub-acute respiratory care covers these patients who require additional administration of steroids.[2,11)]

Summary

Respiratory disorders are the major causes of morbidity and mortality following natural disasters. This presentation described the characteristics of "tsunami lung", which is collectively used for patients with lung lesions and respiratory symptoms due to tsunami. "Tsunami lung" may be considered as a combination of chemical-induced pneumonia and infectious pneumonia caused by bacteria and fungi. Moreover, tsunami causes not only direct pulmonary complications, such as aspiration pneumonia, but also secondary complications through the inhalation of airborne particles from the sludge and rubble. It is important to provide adequate respiratory care for the patients with "tsunami lung", as their condition may be critical without appropriate management.

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RESPIRATORY CARE: TSUNAMI AND FLOOD

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ABSTRACT

The causes of morbidity and mortality due to infection in the population survived from tsunami mainly because of the aspiration of tsunami water caused abnormalities in the lower respiratory tract, either directly or indirectly. *Tsunami* lung is a lung disorder that is associated with a tsunami-related effect on the lungs, including consequences of chemicals aspiration and also bacterial pneumonia. The direct manifestations of the *tsunami lung* depends on the severity in the form of an acute respiratory tract infection, pneumonia, pulmonary edema and respiratory failure. Indirect manifestations can occur after one year due to dust inhalation of the tsunami material residues that contains organic or inorganic particles, which results in a decrease in lung function. Treatment in the acute phase should refer to the etiology and clinical manifestations, but the use of fiber optic bronchoscopy is an alternative. Handling indirect manifestations due to decreased lung function is carried out symptomatically and requires continuous evaluation.

INTRODUCTION

Areas that are demographically described as The Pacific *"ring of fire",* a meeting of the Earth's plate surrounded by an active volcanoes path and the most active earthquake in the world, are extremely vulnerable and high-risk due to the frequent earthquakes and tsunami. Indonesia, as a part of the Pacific *ring of fire*, experienced 19 natural disasters in 2017 and 23 natural disasters in 2018 including earthquake and tsunami in Donggala-Palu and tsunami in Sunda strait. Tsunami impacts mainly on breathing disorders is because of drowning and swept away in the tsunami current. Apart from that, tsunami also causing physical injuries including lung, head injury, fractures, injuries, and water-borne diseases. The main infection problems after the earthquake and tsunami is wound infection and pneumonia. Severe pneumonia caused by the tsunami is known as *tsunami lung* and it is a major infectious in tsunami survivors.

LUNG PROBLEMS DUE TO TSUNAMI

Up to 65% of the main causes of morbidity and mortality in residents who survived the 2005 tsunami disaster in Aceh and Southeast Asia were caused by disorders in the lower respiratory tract, both directly or indirectly. According to the World Health Organization (WHO) after tsunami in Aceh Province-Indonesia in December 2005, based on surveillance and responses toward epidemic diseases data, there were 62% of patients with acute respiratory infections who were very vulnerable. Up to 40% suffered from aspiration pneumonia due to the aspiration of salt water or mud, causing acute respiratory distress syndromes, whereas 2% had another acute complications related to the impact of drowning or swept away. Another studies in Japan found the rate of hospitalization due to pneumonia increased by 5.7 times after 3.5 months after the Japan earthquake and tsunami in 2011 (5). Shibata (2016) found that in the coastal municipalities the death rate due to pneumonia had doubled at 4 weeks post tsunami, the increase occurred between week 1 and week 12. Victims who survived the sinking of the floods and tsunamis experienced aspiration of sea water, mud, and sea debris into the respiratory tract causing inoculum of the infection pathogens in the lungs, which caused pneumonitis and pneumonia. Tsunami and flood victims not experienced water

aspiration only, but also mud soil and other particles carried by water currents causing ARDS, cardiovascular disorders, and cardiac arrest.

Near drowning condition causes respiratory and cardiogenic shock are common problems found in patients affected by the tsunami. Aspiration Pneumonia are generally found a few days after the tsunami among survivors. Patients, who survived a *near drowning* in tsunami, experience aspiration of fluid, sea water and soil debris into the respiratory tract, and it leads to the inoculation of bacteria in the lower respiratory tract. Pathologically, these patients have a necrotic and cavity in their lungs as well another complications such as empyema, pneumothorax, and haematogenous infection into the central nervous system causes neurological complaints.

Aspiration Pneumonia is a direct consequence of a tsunami on the lungs, resulting in infection, reduced alveoli surfactant, pulmonary edema to ARDS, and also non-specific clinical manifestations that range from normal to respiratory failure. Management of this problems is depends on complaints and the severity of the disorder, and it should starts with symptomatic administration, causative antibiotics to invasive treatments including the use of fiber optic bronchoscopy.

Infection in *near drowning* cases is often caused by aerobic gram-negative bacteria, Pseudomonas, Streptococcus Pneumonia, Staphylococcus aureus and anaerobes bacteria. These infection occurs due to the aspiration in oropharynx and also fungi manifestation that occur up to several weeks after aspiration due to tsunami *near drowning.* The rarely found etiology of pneumonia due to aspiration in tsunami, in the Acquired pneumonia community, is especially caused by Burkholderia psedomallei, Escherichia coli and Legionella Pneumonia.

In the 2005 Southeast Asian Tsunami as much as 65% of morbidity and mortality of patients who came to the outpatient clinic associated with *near drowning* and pneumonia, which in longer terms can further develop into pneumothorax, pneumomediastinum and ARDS. Acute Respiratory Infection is the leading cause of death in the first 3-5 days after the tsunami, which in the case of aspiration pneumonia the late diagnosis of prognosis will cause a worse prognostic.

Shibata (2016) found out some variables that contribute in pneumonia due to tsunami includes the lack of nutrition and medication, mental stress and the weather factor. Therefore, in patients with pneumonia attributed by the Tsunami, treatment and intensive supervision is necessary to reduce the risk of death.

In addition to clinical manifestations that depend on the severity, a diffuse bilateral infiltrate as well as the possibility of atelectasis can be found through chest radiography and computed tomography examinations.

In aspiration pneumonia caused by tsunami, the cough reflex is often not sufficient to clean the lower respiratory tract, a bronchoscopy examination should be taken if it is possible as diagnostic and also therapeutic management. Fiber optic bronchoscopy for clearing the respiratory tract (bronchial washing or bronchial toilette) from aspirated materials should be executed by using sterile liquid repeatedly, besides this procedure allows the cultivation of specimens for microorganism culture examination. In some cases earlier fiber optic bronchoscopy treatment can provide better recovery results.

Post-tsunami pulmonary disorders are not only results in direct consequences such as trauma, drowning or aspiration pneumonia, but also results in secondary consequences such as mud particle inhalation and

other particles from materials made from rubber. The inhalation of tsunami mud containing organic and inorganic materials resulting the *Organizing Pneumonia* (OP), a histopathology condition with polyps of intra luminal collagen with fibrotic condition at distal interstitial section, is not understood well yet rarely diagnosed. OP results in nonspecific signs and symptoms, which clinically characterized by progressive productive cough complaints, occurs usually 2-3 weeks after the tsunami because of the inhalation of mud residue particles due to environment cleaning activities while not using personal protection equipment.

Yamanda et al (2016) found several cases of OP in workers involved in post-quake restoration and Japan tsunami in 2011, patients seeks medical treatment with clinical complaints of productive cough and progressive shortness of breath, the diagnosis of OP based on histopathology examination associated with the inhalation of tsunami silt containing inorganic and organic materials.

Acute phase management of pulmonary disorders due to tsunami treated based on the severity and causes, and should be considering several points include:

1. Adequate ventilation with smooth air circulation.
2. Separation of treatment places between patients who have a risk of droplet transmission with patients without risk.
3. Restriction of direct contact between infected and healthy people.
4. Distance between patient's beds should be more than 1 meter.
5. Cleaning and disinfecting of equipment

In the long run, the decrease in lung function occurs not only due to tsunami water aspiration, but it is also associated with dust exposure due to inhalation of tsunami mud residues. A study by Shiga et al conducted 2 years after tsunami found the decline in lung function FEV1 and FEV% in the group who survived the tsunami, compared with those who's not experiencing a tsunami. Decline in lung function occurs due to blockage of peripheral airways, as well as due to fibrosis of the bronchial walls, alveolar walls and interstitium.

To reduce risk, people who carry out activities in areas that have experienced tsunami are required to anticipate the use of masks for personal protective equipment, as well as regular lung function examination. In areas that are demographically described as high risk areas of earthquakes and tsunami, readiness for possibilities that can occur in the lower respiratory tract is a necessity in pre and post-disaster mitigation.

SUMMARY

Tsunami lung is a lung disorder associated with the effects of a tsunami on the lungs, including aspiration of chemicals and bacterial pneumonia caused by the *tsunami*. Pneumonia risk and death caused by *tsunami lung increased* up to two-fold, especially in the coastal areas of society. Apart from the direct result, the long-term *tsunami lung* can be a decrease in lung function. In areas with high risk of tsunami, anticipation of pre and post-disaster needs to be anticipated against complications arising from the lower respiratory system.

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RESPIRATORY CARE ON EARTHQUAKE & LANDSLIDE

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ABSTRACT

Earthquakes and associates tsunamis cause not only direct pulmonary complications such as chest trauma, drowning, or aspiration pneumonia, but also secondary complications via the inhalation of airborne particles from the sludge and rubble. All survivors of disasters are at increased risk of pulmonary disease. Such lung problems may occur as a direct result of the disaster itself, such as inhalation of tsunami water or volcanic dust, or may occur as a result of the post- disaster situation, for example, overcrowding leading to respiratory infections.

THE DIRECT EFFECTS OF NATURAL DISASTERS ON THE LUNG

Many pulmonary complications that occur following natural disasters are a direct result of the disaster itself. The mechanism of insult to the lung as a con- sequence of a natural disaster will vary depending on the nature of events, but in broad terms can be considered under the following categories

- Inhalation of respirable particles, smoke or other toxic gases
- Aspiration of water and water borne pathogens
- Direct trauma to the chest
- Psychological effects causing respiratory symptoms

Earthquakes

Earthquakes are important mainly for the injuries they cause. The spectrum of injuries seen in those with chest trauma includes rib fractures (17-50%); lung collapse (6-52%), and serious bleeding into the chest cavity (11-19%). But cement dust from broken buildings and fungal spores released in air by the rubbles, especially during efforts can cause various diseases. Coccidioidomycosis was the fungal infection with both pulmonary and extra pulmonary diseases in the 1994 Northridge **earthquake** in California.

Tsunamis

Tsunamis are notorious for water borne diseases like cholera and severe injuries. But, after the 2004 Tsunami in Asia, doctors found a new disease called Tsunami lung. Tsunami lung occurs when people being swept by tsunami waves inhale salt-water contaminated with mud and bacteria. The resulting pneumonia-like infections normally are treated with antibiotics. A combination of microbes likely contributes to tsunami lung. However, in a letter published in the 4 April 2005 issue of *The Medical Journal of Australia*, Anthony Allworth, director of infectious diseases at Royal Brisbane and Women's Hospital, describes culturing *Burkholderia pseudomallei* from two tsunami lung patients in a land-based hospital and Nocardia species from a third.

A diagnosis of tsunami lung is based on chest X-ray plus computed tomographic scanning of the brain to document abscesses. A case report published last June described successful antibiotic treatment of a 17-year-old girl who'd lost speech and was partially paralyzed because of brain abscesses. This is shown in the presentation.

Aspiration/near drowning

A sudden rise in water levels, such as during a tsunami, hurricane or a flash flood, is more likely to directly lead to drowning, aspiration and traumatic injury than a more gradual or predictable rise.33 Both occurrences can, however, cause massive disruption of infrastructure, sanitation and population displacement, with attendant health consequences in the medium to longer term.

Aspiration of water into the lung can lead to the introduction of infection, loss of alveolar surfactant, pulmonary edema and ARDS. Pulmonary edema is more common in salt water immersion than fresh water. In addition, vomiting of swallowed water can lead to the aspiration of gastric contents, especially if consciousness and airway protective reflexes are impaired. Signs of significant aspiration are usually detectable clinically, such that those with no signs of aspiration on presentation—no coughing, normal examination, normal blood gases and normal CXR— have a very low likelihood of developing pulmonary edema or pneumonia and are unlikely to require further medical intervention.

Following the 2004 tsunami, near drownings and trauma constituted most of the immediate post- disaster morbidity.36 One medical team reported on 37 patients who had aspirated soil-contaminated salt water. Around half of these developed aspiration pneumonia and eight patients developed ARDS. Pneumothorax (19%) and pneumomediastinum (8%) also occurred as a later complication in those receiving ventilatory support.

The infective sequelae of near drowning reflect the microbial flora of the aspirated water. Different organisms may predominate in freshwater and salt- water aspirations, but aerobic Gram-negative bacteria, including pseudomonas and pseudomonas-like species, are often reported. In addition, colonizers of the oropharynx, such as streptococcus pneumoniae, staphylococcus aureus and anaerobes, may translocate to the lung during aspiration and cause infection. In one series, the majority of organisms isolated from blood or sputum culture of victims of the 2004 tsunami were Gram-negative bacteria, and included cases of Burkholderia pseudomallei, which causes melioidosis and is endemic in South-East Asia. As such, antibiotic therapy may need to include agents active against pseudomonas and any locally prevalent organisms. Antibiotics should be instituted in patients with fever, pulmonary infiltrates and/or signs of systemic toxicity. Fungal infection can also complicate near drowning and should be considered in patients not responding to antibacterial therapy, in those who develop pneumonia some time after the acute aspiration and in those who develop brain abscess or meningitis. Both pseudallescheria boydii and aspergillus species have been reported in this situation.

Respiratory infections, alongside enteric infections, also predominate in the weeks following a flood or tsunami,reflecting a number of factors in addition to direct water borne carriage of pathogens, including population displacement, overcrowding and poor nutritional status. In addition, moulds may contaminate wet buildings following hurricane or flood damage, causing respiratory illness in susceptible individuals.

Natural Disaster and NTM

Infectious diseases acquired by survivors of large-scale natural disasters complicate the recovery process. During events such as tsunamis, hurricanes, earthquakes, and tornados and well into the recovery period, victims often are exposed to water-soil mixtures that have relocated with indigenous microbes. Because nontuberculous mycobacteria (NTM) are ubiquitous in water and soil, there is potential for increased exposure to these organisms during natural disasters. In this hypothesis-driven commentary, we discuss the rise in NTM lung disease and natural disasters and examine the geographic overlap of NTM infections

and disaster frequencies in the United States. Moreover, there has been an increased number of positive NTM cultures from Louisiana residents in the years following three of the relatively recent epic hurricanes and posit that such natural disasters may help to drive the increased number of NTM infections

Current Knowledge of Natural Disaster-Associated NTM Infections

NTM have been recovered from aerosols generated by rivers, dust formed by airflow across rivers, and agricultural fields. During certain natural disasters, there is large-scale mixing of ocean water with fresh water as well as water with soil that likely results in aerosolized NTM and an increased number of NTM in potable and nonpotable water, which may then be inadvertently inhaled and aspirated by survivors. Natural disasters may also displace free-living amoebae from various water niches. Because free-living amoebae can provide an intracellular niche for the NTM to multiply and perhaps become more virulent, development of new microbial symbiosis following natural disasters could potentiate NTM survival and proliferation. Thus, natural disaster survivors may be at increased risk for NTM lung infections resulting from inhalation or aspiration of contaminated water, soil, or NTM-infected amoebae.

Hoefsloot et al found that human respiratory samples from various countries on several continents show diverse species of NTM, with *Mycobacterium avium complex* being the most frequently recovered, yet there are few published reports of NTM infections acquired after natural disasters.

Potential mechanisms by which natural disasters could increase NTM lung infections are increased aspiration and inhalation of NTM-contaminated water and aero- sols, increased NTM inoculum size resulting from the disrupted environment, and increased susceptibility due to immunosuppression from malnutrition, sleep depri- vation, and other physical and emotional stressors. Other mechanisms include more time spent outdoors by individuals living in several of the high NTM-prevalent states with warmer climates as well as an increased pro- portion of retirees residing in many of these states, who because of their older age, are more susceptible to NTM lung disease.

A hypotheses regarding the potential link between natural disasters and NTM infections:

1. NTM infections are more likely to occur in survivors following natural disasters as a result of the disruption of water and soil ecosystems normally inhabited by NTM;
2. The retirement communities of Louisiana, Florida, California, and Hawaii may be disproportionately affected given the frequency of natural disasters in these areas and the increased vulnerability of elderly people to pulmonary NTM infections;
3. The risk of NTM infection may remain for weeks, months, or years following a natural disaster due to further disruption of the ecosystem during the reconstruction periods; and
4. NTM lung disease may not manifest or be diagnosed for months or years after the initial infection and, thus, the initial link to natural disasters may not be recognized.

Long-term, prospective microbiologic and epidemiology studies in disaster-prone areas are needed to validate this hypothesis that NTM lung infections are linked to natural disasters. These studies should include reliable documentation of whether a positive NTM culture is due to an environmental contaminant, a colonizer, or a true infection. This hypothesis- driven discussion of a potential and perhaps underappreciated public health issue: natural disaster-associated pulmonary NTM infections. The upward trend in both NTM infections and natural disasters emphasizes the need to confirm whether these “impending storms” are linked.

Two cases of Tsunami Dust Pneumonia are presented. These two cases had organizing pneumonia (OP) secondary to the inhalation of the dried tsunami sludge which formed during the 2011 Great East Japan Earthquake and the consequent tsunami. After the disaster, both of these patients had been engaged in the restoration work. Without protective gear. About half a month later, they developed shortness of breath and pulmonary infiltrates. These patients were diagnosed with interstitial pneumonia. Their biopsy specimens revealed multifocal peribronchiolitis and OP. An electron probe microanalysis of these specimens demonstrated the presence of elements from the earth's crust in the inflammatory lesions. These two cases indicate that exposure to dried tsunami sludge can cause OP.

The dried sludge that these two patients inhaled was composed primarily of earth and sand from the seabed, in addition to microorganisms, organic compounds, oils, heavy metals, and chemical compounds such as dioxins and poly- vinyl chloride. We did not detect any particular cause of OP and believed that the size and concentration of the dust had most likely strongly influenced the development of OP. An elemental analysis primarily indicated the presence of Fe, Si, Al, and Ca, with large amounts of oxygen in the peribronchioles and alveolar walls; however, no other elements were found. The size of these earth elements in the atmosphere is usually 5 to 30 micrometers and most of them become trapped in the upper respiratory tract, but only those smaller than 5 micrometers reach the bronchioles and alveoli . A study on the constitution of the tsunami sludge indicated the presence of approximately 10% to 80 % of silt and clay in the elements with a size of less than 7.5 micrometers. Therefore, the dried sludge from the tsunami can reach the peripheral zone of the lung. In the Hanshin-Awaji earth- quake, the damaged area presented high concentrations of airborne particles, particularly on sunny days . The authors believe that the patients were exposed to high concentrations of dust because most of their work had been indoors. It is also important to note that our patients did not use any counter- measures against dust, such as wearing a dust protective mask.

These cases emphasize the urgent need to protect individuals who engage in restoration work from dust inhalation in the aftermath of a tsunami or other similar natural disaster.

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VOLCANO ERUPTION AND FIRE

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ABSTRACT

Introduction

There are a number of active volcanoes throughout the world and many of them are close to urban settings and major cities. At times, they are responsible for pyroclastic emissions, consisting of a mixture of gases, vapours, aerosols, and particulate matter. These emissions have the potential to cause massive environmental pollution and impact on climate, surface infra structures and human activities, thus resulting in a significant economic burden for both the community and the individuals.[1] Among the adverse effects of pyroclastic emissions (ash fall), those related to human health raise significant concern in both exposed populations and health service administrators. [1,2]

Volcanic Emission

The type and physicochemical characteristics of volcanic emissions vary depending on the volcano. In fact, these characteristics and types depend on the volcanic morphology and the geological features of the region where the volcano is located. Generally, volcanic ashes are composed of magmatic fragments, consisting of both glass and minerals.

The mineralogical composition of volcanic ash consists of approximately 45–75wt% silica (SiO2), making SiO2 content useful as a classification parameter.3 There are also other major components in volcanic emissions, such as water vapor, hydrogen peroxide (H2O2), carbon dioxide (CO2), sulfur dioxide (SO2) (the dominant sulfur component), hydrogen sulphide (H2S) (the second most important S species, converted to SO2 in the atmosphere), sulfates (SO4), and carbonyl sulfide (COS) and its precursor carbon disulfide (CS2).

Carbonyl sulphide (COS) has a residence time of several years in the atmosphere and is an important source of sulfate aerosols. The main halogen component of volcanic ash is hydrogen chloride (HCl), which is highly soluble and is rapidly washed out from the atmosphere. Hydrogen fluoride (HF), which is another ash component, may be dangerous because it is introduced into the alimentary chain mainly through contaminated water, not to mention that fluoride is an emergent toxicant affecting smooth organs (e.g., lungs and kidney).[4] Volcanic ash is a source of helium (He), radon (Rn), mercury (Hg), magnesium (Mg), manganese (Mn) and bromide (Br), increasing the risk for people who have difficulty in absorbing essential elements such as Ca, Fe and Zn and retaining other elements such as Cd and Mn.[5]

The 2010 eruptions of Merapi Volcano began in late October 2010 when this volcano in Central Java, Indonesia began an increasingly violent series of eruptions that continued into November 2010. Among parts of materials ejected by Merapi Volcano, volcanic ash may cause acute respiratory diseases (e.g. asthma and bronchitis) and has the potential to instigate chronic diseases such as silicosis and lung cancer. Volcanic ash is considered a respiratory health hazard because of several potentially toxic components, such as respirable crystalline silica. Volcanic ash can carry a variety of potentially toxic adsorbed elements such as Cl, S, Na, Ca, K, Mg and F as well as metals such as Pb, Hg, Cu, Zn, Cd and As and other potentially pathogenic trace elements. These elements may be carried hundreds of kilometres in a volcanic plume and will be inhaled with the ash particles unless leaching occurs first.[6]

Respiratory Health Effect

Humans experience multiple types of exposure (respiratory and gastrointestinal tract, skin contact) to volcanic emissions. The respiratory system is the most susceptible because of the large quantities of air we breathe (20 m3/day). Moreover, the impact of such exposure depends on different factors; essentially, the physical and chemical characteristics of the toxic substances (e.g., heavy metals, salts, metal oxides, inorganic carbons, silicates, plastics or organics) are in question. In addition, the age, sex, respiratory pattern and health status of the exposed person are also determinant factors. Among the physical characteristics, the size of the ash particles emitted by volcanoes, which can be less than 2 mm , and the number, concentration and density of the particles as well as the dynamics of the gas flow in the airways will determine the region where the ash particles will be deposited and will determine the local adverse effects.[7]

The health effects of the volcanic emissions depend on the physical and chemical characteristics of the emissions and on the corresponding toxicological properties. The health effects are a consequence of the inhalation of particles directly emitted from active volcanoes or of resuspension of the soil ashes during the cleanup after the eruption. The effects that such exposure has on human health can be classified as acute or chronic. Acute effects include eye and throat irritation, cough, dyspnea, wheezing, chronic obstructive pulmonary disease (COPD), cardiovascular events, psychological stress, reversible changes in healthy lung function, and acute exacerbations of previously existing respiratory conditions such as asthma.[8,9] As the lung is not a closed system, tiny particles (<0.1 μm) called ultrafine particles (UFPs) can translocate to other organs, where these particles can have adverse effects.[7]

Components of the ash particles can be dissolved in the lung-lining fluid and can pass through the alveolar-capillary membrane; this interaction explains, at least in part, the relationship between particle exposure, cardiovascular disease and some neurological alterations.[7] Chronic effects are related to increased mortality rate for cardiopulmonary disease,[9] increased medication use (e.g., asthma medication, analgesics),[11] and increased prevalence of some types of cancer (e.g., lip, oral cavity, pharynx and female breast cancers).[12] Moreover, neurodegenerative diseases such as Alzheimer's disease have also been linked to chronic exposure to volcanic emissions.[13] Animal models have reported that volcanic emissions are associated with impairment of spermatogenesis.[14]

Longo et al. found a statistically significant positive association between chronic exposure to SO2 and fine sulfate particles (PM2.5) emitted from the Kilauea volcano (Hawai'i) and increased prevalence of cough, phlegm, rhinorrhea, sore/dry throat, sinus congestion, wheezing, eye irritation and bronchitis.[15] The chronic exposure to volcanic SO2 emissions increased the risk of acute bronchitis in children aged 0–14 years, with a cumulative incidence ratio of 6.56.[15] These findings are consistent with the hypothesis that short exposure to volcanic ash is associated with reversible inflammation of the airways.[16]

Tam et al. found that chronic exposure to respirable acidic particulates is associated with a reduction in the FEV1/FVC ratio; however, this effect was not statistically significant. The authors did not find any association with the diagnosis of asthma or with persistent wheezing or bronchitis in the last 12 months in the group studied.[17] Previous observations report that although volcanic ash may not be acutely toxic, its exposure has been associated with a variety of health effects - spanning from sudden, asphyxia-induced death due to acute respiratory tract irritation and symptoms either in subjects with existing respiratory disorders like asthma or healthy individuals.[18,19,20,21]

Mt. Sakurajima in Japan is one of the most active volcanoes in the world. In the last century, the volcano became active every 10–30 years. Its volcanic ash contains up to 7wt.% of cristobalite, which is known to be carcinogenic. However, the chronic health effects of Mt. Sakurajima's volcanic activities were not clear.[22]

Study in Iceland by Carlsen et al documented a high prevalence of respiratory symptoms 6–9 months following the volcanic eruption in Eyjafjallajökull, especially among those most exposed. Also, subgroups who reported more than one physical symptom were more prone to experience psychological difficulties. The study reveals that the adverse health effects of a volcanic eruption may last for many months beyond the eruption and the immediate disaster relief services provided. This is important for health authorities to bear in mind.[23] Gudmundsson in his study found that acute respiratory symptoms after exposure to volcanic ash are well described but no long-term effects have been found.[24]

Mortality

Study in Japan shown that Mt. Sakurajima's volcanic activities increased the mortality of lung cancer and COPDs in the Sakurajima-Tarumizu area, which has the largest amount of ashfall from the volcano in its vicinity.[22]

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COMMUNITY ACQUIRED PNEUMONIA, FROM MODERATE TO SEVERE

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ABSTRACT

Community-acquired pneumonia (CAP) is defined as an acute symptomatic infection of the lower respiratory tract in patients outside a hospital or a long-term care facility, whereby a new infiltrate is demonstrated.1,2 CAP is a common condition that carries a high burden of mortality and morbidity, particularly in the elderly.

The manifestations of community-acquired pneumonia include respiratory symptoms (cough, sputum, dys - pnea, chest pain) and general symptoms of infection (fever, hypothermia, malaise, flu-like symptoms, circulatory symptoms, impaired consciousness), along with the corresponding physical findings (tachypnea, tachycardia, arterial hypotension, focal auscultatory abnormality). As these manifestations are not sensitive or specific enough for definitive diagnosis (e1), a confirmatory chest x-ray is recommended. Infiltrates can also be detected by chest ultrasonography.

Patients with CAP may be classified according to severity: I) mild, II) moderately severe, III) severe CAP admitted to the ward and IV) severe CAP admitted to the intensive care unit (ICU). Two validated scoring systems are in use: the Pneumonia Severity Index and the CURB-65. Alternatively, a pragmatic classification (treatment at home; admission to a general medical ward and admission to ICU) can be used. Selection of empirical antibiotic therapy should be guided by the severity of the illness at presentation, site of care, and most likely pathogens. Start antibiotics as soon as we are confident that CAP is the appropriate working diagnosis and, ideally, within four hours of presentation for inpatients and within one hour of presentation for those who are critically ill.

Community-acquired pneumonia (CAP) is defined as an acute symptomatic infection of the lower respiratory tract in patients outside a hospital or a long-term care facility, whereby a new infiltrate is demonstrated. CAP is a common condition that carries a high burden of mortality and morbidity, particularly in the elderly.[1,2]

Incidence

CAP is one of the most common and morbid conditions encountered in clinical practice. In the United States, CAP accounts for over 4.5 million outpatient and emergency room visits annually, corresponding to approximately 0.4 percent of all encounters. CAP is the second most common cause of hospitalization and the most common infectious cause of death. Approximately 650 adults are hospitalized with CAP every year per 100,000 population in the United States, corresponding to 1.5 million unique CAP hospitalizations each year. Nearly 9 percent of patients hospitalized with CAP will be rehospitalized due to a new episode of CAP during the same year.[3,4]

In Indonesia pneumonia is one of the ten most common inpatient disease in hospital corresponding to 53,95% of men and 46,05% of women, with crude fatality rate (CFR) 7,6% highest level according to another disease.[5]

RISK FACTORS

Include age ≥65 years, chronic comorbidities, concurrent or antecedent respiratory viral infections, impaired airway protection, smoking, alcohol abuse, and other lifestyle factors (eg, crowded living conditions)[6]

PATHOGENESIS1-3

Traditionally, CAP has been viewed as an infection of the lung parenchyma, primarily caused by bacterial or viral respiratory pathogens. In this model, respiratory pathogens are transmitted from person to person via droplets or, less commonly, via aerosol inhalation (eg, as with *Legionella or Coxiella species*). Following inhalation, the pathogen colonizes the nasopharynx and then reaches the lung alveoli via microaspiration. When the inoculum size is sufficient and/or host immune defenses are impaired, infection results. Replication of the pathogen, the production of virulence factors, and the host immune response lead to inflammation and damage of the lung parenchyma, resulting in pneumonia

In some cases, CAP might also arise from uncontrolled replication of microbes that normally reside in the alveoli. The alveolar microbiome is similar to oral flora and primarily comprised of anaerobic bacteria (eg, *Prevotella and Veillonella*) and microaerophilic streptococci. Hypothetically, exogenous insults such as a viral infections or smoke exposure might alter the composition of the alveolar microbiome and trigger overgrowth of certain microbes. Because organisms that comprise the alveolar microbiome typically cannot be cultivated using standard cultures, this hypothesis might explain the low rate of pathogen detection among patients with CAP.

In any scenario, the host immune response to microbial replication within the alveoli plays an important role in determining disease severity. For some patients, a local inflammatory response within the lung predominate and be sufficient for controlling infection. In others, a systemic response is necessary to control infection and prevent spread or complications, such as bacteremia. In a minority, the systemic response can become dysregulated, leading to tissue injury, sepsis, acute respiratory distress syndrome, and/or multiorgan dysfunction.

DIAGNOSTIC EVALUATION

The manifestations of community-acquired pneumonia include respiratory symptoms (cough, sputum, dyspnea, chest pain) and general symptoms of infection (fever, hypothermia, malaise, flu-like symptoms, circulatory symptoms, impaired consciousness), along with the corresponding physical findings (tachypnea, tachycardia, arterial hypotension, focal auscultatory abnormality). As these manifestations are not sensitive or specific enough for definitive diagnosis, a confirmatory chest x-ray is recommended. Infiltrates can also be detected by chest ultrasonography. The following clinical findings elevate the pretest probability that an infiltrate will be present and should prompt a chest x-ray, also in the outpatient setting. [2,4,6]

Patients with CAP may be classified according to severity:[1-3]

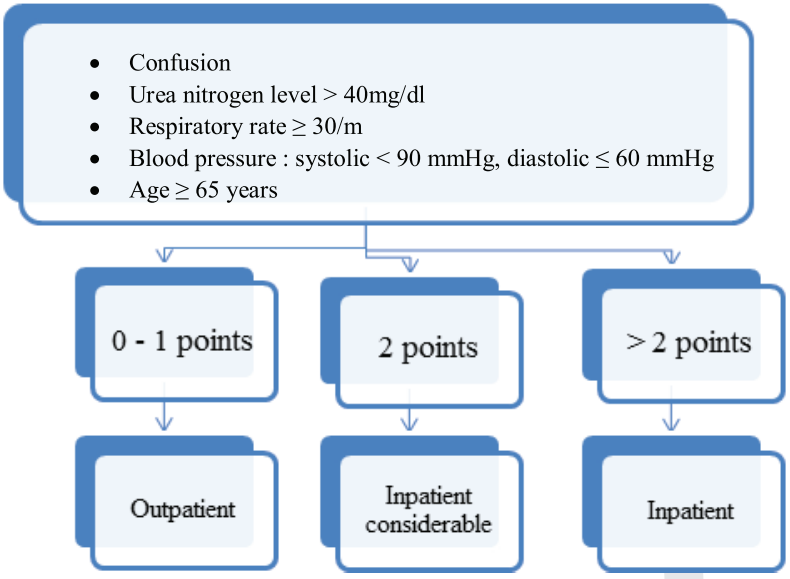
I) mild, II) moderately severe, III) severe CAP admitted to the ward and IV) severe CAP admitted to the intensive care unit (ICU).

Two validated scoring systems are in use: the Pneumonia Severity Index (PSI) and the CURB-65. Alternatively, a pragmatic classification (treatment at home; admission to a general medical ward and admission to ICU) can be used. It's not recommend any of these scoring systems over the others; however, we recommend that each hospital uses only one scoring system consistently in daily practice.

The most commonly used severity scores are the Pneumonia Severity Index (PSI) and CURB-65. We generally prefer the PSI, also known as the PORT score because it is the most accurate and its safety and effectiveness in guiding clinical decision making have been validated. However, the CURB-65 score is a reasonable alternative and preferred by many clinicians because it is easier to use. [2,3]

For patients with risk category III (severe CAP – ward admission; CURB-65: 3-5; hospitalised on non-ICU ward) therapy should be started with a 2nd or 3rd generation cephalosporin. No empirical coverage for atypical microorganisms is given. A Legionella and pneumococcal urinary antigen test should be carried out as a routine procedure within 12-24 hours of admission. If the Legionella test is positive, monotherapy directed against Legionella spp. is recommended. If the pneumococcal urinary antigen test is positive, therapy can be narrowed to penicillin or amoxicillin. If both are negative, therapy is continued with a 2nd or 3rd generation cephalosporin, to provide additional coverage for *Enterobacteriaceae* and to a lesser extent S. aureus.[2,7,8]

For patients with category IV (severe CAP – ICU admission; hospitalised on ICU ward) it is always recommended to cover *S. pneumoniae,* Legionella spp. and Gram-negative infections. For this purpose there are two equally acceptable choices, both with excellent antimicrobial activity against all expected causative agents: (a) monotherapy with moxifloxacin or (b) combination therapy with a 2nd or 3rd generation cephalosporin and ciprofloxacin. Macrolides are no longer recommended in this patient category. For all patients in category IV, a Legionella urinary antigen and S. *pneumoniae* urine antigen test is carried out as a routine procedure within 12-24 hours of admission. If the Legionella test is positive, monotherapy directed against Legionella spp. is recommended. If the Legionella test is negative, the patient is still treated further with combination therapy (coverage of both S. *pneumoniae* and *Legionella* spp.) because the sensitivity of the urinary antigen test is not 100%. Since the specificity of the pneumococcal urine antigen test is < 100%, antibiotic treatment can be streamlined to penicillin or amoxicillin only in patients with a positive test result and without another pathogen detected once clinical stability (often within 48 hours) has been reached.



Corticosteroids are not recommended as adjunctive therapy for treatment of CAP.[2,7,8]

CURB-65 Mortality Prediction Tools for Patients with Community-Acquired Pneumonia consists:

- Score 0 – 1 : low mortality risk, patients may outpatient
- Score 2 : average mortality risk, patients considerable inpatient
- Score > 3 : high mortality risk, patients considered as severe pneumonia and must be inpatient
- Score 4 or 5 : patients must be considered admitted to intensive care unit

Adapted from 5

Demographic Factor	Score
Age: Men	Age in years
Women	Age –10
Nursing Home Resident	Age + 10
Coexisting Illnesses	
Neoplastic disease	+30
Liver Disease	+20
Congestive heart failure	+10
Cerebrovascular Disease	+10
Renal Disease	+10
Physical Examination Findings	
Altered Mental Status	+20
Respiratory Rate >30	+20
Systolic Blood Pressure <90 mmHg	+20
Temperature < 35 or > 40° C	+15
Pulse > 125/min	+10
Laboratory and Radiographic Findings	
Arterial pH < 7.35	+30
BUN > 30 mg/dl	+20
Sodium <130 mEq/L	+20
Glucose >250 mg/dl	+10
Hematocrit < 30%	+10
Partial pressure of arterial oxygen < 60mmHg	+10
Pleural effusion	+10
TOTAL SCORE	

PSI Scoring System[9]

Patient Score	Risk level	Class	Mortality risk	Treatment Setting
Unpredicted	Low	Class I	0,1%	Outpatient
51- 70		Class II	0,6%	Outpatient
71-90		Class III	2,8%	Overnight admission
91-130	Average	Class IV	8,2%	Hospital Unit
>130	Severe	Class V	29,2%	ICU

Treatment setting decision based on PSI score[9]

Bundle of measures for severe community-acquired pneumonia

To be performed as rapidly as possible within the first 3 hours:[1]

- Serum lactate measurement
- Blood cultures
- Initiation of appropriate (generally combined) broad-spectrum intravenous antibiotic treatment, within one hour if possible
- In patients with arterial hypotension or elevated serum lactate, rapid intra -venous administration of crystalloids
- Evaluation (including blood-gas analysis) and treatment, if needed, of acute respiratory insufficiency

To be performed within the first 6 hours:[1]

- Administration of vasopressors if the response to volume administration is inadequate
- Repetition of lactate measurement if the initial value was elevated
- Re-evaluation of blood gases

Severity Class	Primary treatment (standart dose)	Alternative treatment (standart dose)
Moderately severe CAP **(no acute organ dysfunction)**	*Beta-lactam IV* – Amoxicillin/clavulanic acid (2.2 g q8h) – Ampicillin/sulbactam (3 g q8h) – Cefuroxime (1.5 g q8h) – Ceftriaxone (2 g qd) – Cefotaxime (2 g q8h) *+ Optional* macrolide IV or po for 3 days* – Clarithromycin (500 mg q12h) – Azithromycin (500 mg qd)	*Fluoroquinolone IV or po* Moxifloxacin (400 mg qd) Levofloxacin (500 mg qd or q12h)
Severe CAP **(acute organ dysfunction)**	*Beta-lactam IV* – Piperacillin/tazobactam (4.5 g q6-8h) – Ceftriaxone (2 g qd) – Cefotaxime (2 g q6-8h) *+ Macrolide IV for 3 days* – Clarithromycin (500 mg q12h) – Azithromycin (500 mg qd)	*Fluoroquinolone IV* Moxifloxacin (400 mg qd) Levofloxacin (500 mg q12h) (no monotherapy in patients with septic shock)

Empirical initial treatment for in-hospital therapy of community-acquired pneumonia.[1]

* The additional administration of a macrolide is optional because prospective, placebo-controlled trials have not clearly shown that they improve the outcome

Criteria for clinical stability.[1]

The signs of clinical stability are defined as:

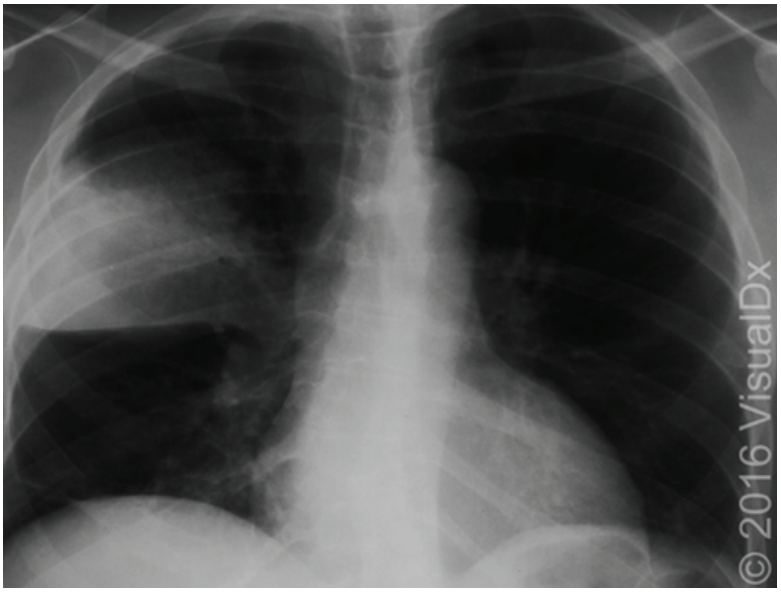
- Heart rate ≤ 100/min
- Respiratory rate ≤ 24/min
- Systolic blood pressure ≥ 90 mm Hg
- Body temperature ≤ 37.8°C
- Ability to take food by mouth
- Normal state of consciousness
- No hypoxemia (pO2 ≥ 60 mmHg, SaO2 ≥ 90%)

pO2, partial pressure of oxygen; SaO2, oxygen saturationAl

For all patients, treated until the patient has been afebrile and clinically stable for at least 48 hours and for a minimum of five days. Patients with mild infection generally require five to seven days of therapy; those with severe infection or chronic comorbidities generally require 7 to 10 days of therapy.
Failure to respond to antibiotic treatment within 72 hours should prompt reconsideration of the diagnosis and empiric treatment regimen as well as an assessment for complications.[1,2,8]

Treatment failure
If clinical stability does not ensue or if there is clinical worsening after 3–5 days of appropriate treatment, the patient must be evaluated for treatment failure. Progressive pneumonia carries an up to tenfold increase in mortality.[3]

Cardiovascular complications
Patients with pre-existing cardiac disease need repeated evaluation for potential cardiac complications and reassessment of the indication for acetylsalicylic acid treatment.[3]
Mortality — Although the majority of patient with CAP recover without complications, CAP is a severe illness and among the leading causes of mortality worldwide. Mortality can be directly attributable to CAP (eg, overwhelming sepsis or respiratory failure) or can result indirectly from cardiovascular events or other comorbid complications (eg, advanced chronic obstructive pulmonary disease [COPD] [2,3]



Chest radiograph showing right upper lobe infiltrated in a patient with pneumonia.[4]

Source: https://emedicine.medscape.com/article/360090-overview

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INVASIVE FUNGAL INFECTION, FOCUS ON PULMONARY MYCOSIS

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Extended Abstract

Fungal infection occurs generally in patients with immunocompromised conditions. Candida and Aspergillus sp are the most common cause of infection in such patients, of which Candida albicans and Aspergillus fumigatus are the most frequent. Other species are Cryptococcus, Histoplasma, Trichosporon, Zygomycetes and Fusarium. Recently, nonalbicans candidosis and non-fumigatus aspergillosis are becoming more frequent and resistant candidas and aspergillus are more frequently found.

As for pulmonary mycosis, the important predisposing factor is the pre-existing lung diseases like tuberculosis, bronchiectasis, COPD, asthma and lung cancer. Pulmonary mycosis may be caused by Aspergillus, Histoplasma, Cryptococcus or Pneumocystis, while pulmonary candidiasis is considerably rare. In patients with serious lung conditions, **candidemia** may develop as a serious condition which may lead to death. Pulmonary cryptococcosis arise frequently in patients with positive HIV or AIDS. Besides the lung, this fungus also affects central nervous system, causing meningitis and encephalitis, both may be fatal. Patients with HIV or AIDS may also develop pneumonia previously believed to be caused by Pneumocysitis carinii, hence called pneumocystis carinii pneumonia, which was not proven to be so, hence the condition is now called pneumocystis pneumonia.

Histoplasmosis of the lung has not yet been well known in Indonesia, a wide research is now being planned in several centers to identify specific conditions atributed to this type of pulmonary mycosis. Occasional cases have occurred in some centers, but the frequency has not yet been established.

Risk Factors for Sistemic Mycosis
Several risk factors play important role in causing systemic fungal infection, including pulmonary mycosis, such as ICU stay, gastrointestinal surgery, total parenteral nutrition, central venous catheter, cytotoxic drugs, broad spectrum antibiotics, steroids, fungal colonization, neutropenia, renal failure, prematurity, malignancies etc.

A study on 363 cases of candidemia in 12 hospitals in Korea shows that prior antibiotic therapy is the most frequent underlying disease (93,4%), followed by central venous catheter (78,4), total parenteral nutition (66,4%), blood transfusion (61,2%), ICU stay (48,5%), malignancy (44%) and neutropenia (11%).
According to Comilet (2006), underlying diseases for invasive aspergillosis are hematologic malignancies (57%), pre-existing pulmonary diseases (19%), solid organ transplantation (11%), systemic diseases, (7%), solid tumors (3%), AIDS (1%) and unknown condition (2%).

Clinical features and Diagnosis
The symptoms and signs of pulmonary mycosis do not differ much from those of other pulmonary infections, especially chronic diseases. Cough, sputum production, shortness of breath, low fever and loss of appetite are main symptoms, which may persist after treatment. In candidemia, high fever may remain in spite of adequate antibiotic treatment. As in symptoms, there is no pathognomonic finding in physical examination, findings may be similar to all infectious disease of the lungs. The same conditions are also seen in clinical

laboratory tests. Leucocytosis is not common for fungal infection, it may show corresponding bacterial infection. The radiologic picture of chest may show infiltrates similar to bacterial pneumonia or tuberculosis. A rounded shadow with **halo sign that develops to be a crescent shadow when followed by CT scanning** may suggest invasive aspergillosis, **ad a rounded shadow inside a clean cavity** may suggest aspergilloma growing in a previous tuberculosis cavity. **Multiple nodules of various sizes** may suggest cryptococcosis or histoplasmosis.

To make the diagnosis of pulmonary mycosis, one should have **strong suspicion** towards the disease, otherwise one tends to forget the possibility. The suspicion towards the infection of fungus must be **based on the risk factors as described above and must be followed by laboratory tests, namely mycologic examination and, if possible, histopathologic examination.** Positive finding for culture of certain fungus or positive histologic finding showing the existence of fungus in the tissue make a **proven diagnosis**. When diagnosis is based on clinical symptoms plus negative mycology tests or the tests have not been performed or the result of which have not been available, such diagnosis is **possible. Probable diagnosis** is based on clinical features, risk factors and positive mycology tests other than histology or culture.

Mycology tests consist of direct examination and culture of sputum or bronchial secretion and serologic tests for antigen or antibody against fungus. The finding of spores and positive culture of Candida in sputum does not automatically confirm the diagnosis of Candidosis, since this fungus can be detected in normal patients as saprophytes. When the findings come from bronchial washing, there is more possibility as the causing agent. Positive finding of **antibody** to Candida or Aspergillus cannot be used as diagnostic determinant since one single cell of the fungi can be recognized by the antibody, even when the fungus is not pathogenic in nature. Parts of hyphae may be seen in sputum or bronchial washing, this may indicate the existence of Aspergillus. When an antigen called galactomannan is detected in the sputum, bronchial washing, or in serum, this may confirm the diagnosis of invasive pulmonary aspergillosis. Galactomannan is an antigen produced by Aspergillus as the fungus invades the tissue into the lung or bronchus. The finding of Cryptococcus and Histoplasma always means positive diseases.

Treatment of Pulmonary Fungal Infection

Treatment of pulmonary mycosis can be prophylactic, empiric, preemptive or causal. **Causal treatment** is given based on the proven existance of fungus either histologically or mycologically (by culture from organ or fluids which are normally sterile). **Prophylactic** treatment is given to cases in which systemic fungal infection most probably will occur after certain treatment or operation, such as organ transplantation. The most common treatment modalities are **empiric or preemptive**. When a patient has one or several risk factors with clinical symptoms and certain radiologic feature which may lead to systemic fungal infection, empiric treatment with antifungal drug can be administered before any mycologic test has been performed or the result of which have not been published. When colonization was found in such patient but no causal agent has been identified, the patient can be treated pre-emptively.

In real world, treatment of systemic fungal infection tends to be delayed, due to late diagnosis, which may result in unsatisfying result. When treatment was delayed for 4 hours, the risk of death can occur 4 times as more frequent. Therefore waiting for histologic prove or positive result of culture may increase the risk of unsuccessful treatment and death, hence probable or possible diagnosis of fungal infection should be followed by antifungal therapy.

There are several drugs for systemic fungal infection, namely **amphotericin B, the azoles and**

echinocandins. Amphotericin B is the most effective drug for all, but it is hardly used at present, due to the severe toxicity towards the kidneys that may lead to renal failure. In good hands, however, if the dose is managed carefully, the toxicity would not be a problem. The dose should be started low, 0,5 mg/kg BW, then gradually increased up to 1 mg/kg BW if no sign of renal toxicity occurs. Nowadays, Amphotericin B is not available on market, in Indonesia it is available for free only for IHV patients.

Echinocandins like mycafungin, anidulafungin and caspofungin are safe drugs for severely ill or moderately ill patients. Anidulafungin and caspofungin need loading dose to start (day 1), followed by maintenance dose, while mycafungin can be used without loading dose. Caspofungin is not available in Indonesia. Mycafungin and anidulafungin should be administ-ered by intravenous drip, 100mg daily for several days then shift to fluconazole 400 mg, when the patient shows improvement. Anidulafungin should be started as a loading dose of 200mg at the first day then continue with 100 mg daily. The loading dose for caspofungin is 70mg, maintenance dose is 50mg. Mycafungin does need a loading dose. Echinocandins are effective against Candida and is save for liver.

Among **azoles**, fluconazole is the most widely used and is generally available. For systemic /pulmonary mycosis, the dose is 12 mg/kg BW as loading dose then continued by 6 mg/kg BW. Fluconazole can be used as continuation therapy after amphotericin or echinocandin treatment, when the patient has shown clinical improvement. Fluconazole is effective against Candida, Histoplasma and Cryptococcus, but not against Aspergillus. For invasive pulmonary aspergillosis, the drug of choice is Voriconazole, 200-400 mg. Other drugs that is effective against aspergillosis is itraconazole and ketokconazole. Azoles should be used with caution in patients with liver function impairment.

The duration of treatment for systemic mycosis varied between 8 weeks to 12 months. Clinical improvement is the most important criteria, while serial serologic test like galactomannan may be used as guide, but it is not always a good criteria to cease the treatment.

Treatment of Candidemia

Critically ill patients with high fever who stays in ICU, with pre-existing lung infection that is not responding to adequate antibiotic treatment should be strongly suspected as candidemia sepsis. An echinocandin (mycafungin, anidulafungin or caspofungin) is the drug of choice for **candidemia sepsis plus neutropenia.** When the patient responds well, the treatment can be shifted to fluconazole. All catheters and indwelling devices should be removed as soon as possible and leucopenia should be improved. Candidemia in less critically ill patients without leucopenia may be treated with intravenous drip of fluconazole, the loading dose is 800mg (12 mg/kg BW) on the 1st day, followed by 400mg (46 mg/kg BW) for 2-4 weeks.

Treatment of Aspergillosis

Invasive pulmonary aspergillosis and chronic pulmonary aspergillosis cannot be treated with fluconazole. Chronic pulmonary aspergillosis is usually found in patients already having chronic pulmonary diseases. The drug of choice for aspergillosis is voriconazole given as intravenous drip or orally, the maximal dose is 4 mg/kg BW. Seriously ill patients should receive intravenous treatment. Alternative drug is amphotercine B deoxycholate (AmB-d), which is now hardly used even if is available, due to severe side effects to the kidneys leading to renal failure. The dose for AmB is 0.5 mg/kg BW daily, carefully increased to 1.0 mg/kg BW, while observing carefully for signs of renal toxicity. When lipid formulation (LFAmB) is used, the dosage is 3-5 mg/kg BW daily. Duration of treatment is up 6-12 weeks and in imuno-supressed condition,

treatment should be continued until the lesion disappears. Mortality rate of aspergillosis is 58% (87% in bone marrow transplantation, 90% in CNS dissemination).

Surgical resection is recommended for patients with aspergilloma, especially when hemo-ptysis occurs and is life threatening. Antifungal treatment only may not be beneficial in such patients.

In allergic bronchopulmonary aspergillosis, the treatment is mainly given for the control of asthma. Antifungal treatment does not play important role, since the fungus grows on the surface of bronchus.

Treatment of Other Pulmonary Mycoses

Histoplasmosis is caused by Histoplasma capsulatum, the most frequent endemic mycosis. It has emerged as the complication of HIV-AIDS. Drug of choice for the treatment of systemic histoplasmosis is amphotericin B 0,7 mg/kg BW, given sequentially with itraconazole 200-400 mg/day for 6-12 months or ketoconazole 400-800 mg/day (ketoconazole is less effective). Another choice for treatment is voriconazole orally, the dose for adults over 40 kg is 6 mg/kg BW every 12 hours, given before meals.

Cryptococcosis is caused by Cryptococcus neoformans, the incidence has risen in connection to HIV-AIDS. The spectrum of disease ranges from asymptomatic pulmonary infection in a normal host to a rapidly fatal disease in immunocompromised individuals. Meningo-encephalitis is the most frequently diagnosed, pulmonary infection maybe discovered in dissemination of disease. Asymptomatic pulmonary cryptococcosis in **HIV negative** patients may be observed for the progression of disease. When mild to moderate symptoms occur, treatment with fluconazole may be given 200-400 mg/day for 6-12 months. When severe symptoms occur, use the regimen for meningitis. Asymptomatic patients with HIV positive may be observed or treated as patients with mild to moderate sympoms as described above, whereas those with severe symptoms must be treated as meningitis.

Treatment of cryptococcal meningitis in HIV negative as well as HIV positive patients consists of amphotericin B 0,7-1 mg/kg BW plus of oral 5-flucytosin 100 mg/kg BW daily for 2 weeks as induction, followed by fluconazole 400 mg/day for 10 weeks.

Pneumocystis pneumonia is treated with trimetrophim-sulfamethoxazole (TMP-SMX) as the first-line agent for the treatment of mild to severe PCP. Empiric treatment should be considered in severe patients who have risk factors and clinical manifestations. All severe cases should be treated intravenously in hospital, oral administration of TMP-SMX is considered in mild cases or after initial improvement. The duration of treatment is generally 3 weeks for patients with HIV, or maybe longer. Some adverse events, such as hepatotoxicity, nephrotoxicity, bone marrow depression, and skin rash, sometimes become obstacle to the completion of the treatment. The recommended daily dose is TMP 15–20 mg/kg plus SMX 75–100 mg/kg, but the optimal dose of TMP-SMX remains unclear. A retrospective investigation by Thomas et al revealed a good outcome with TMP 10 mg/kg/day plus SMX 50 mg/kg/day for PCP in HIV-infected patients.

Summary

For pulmonary mycosis, the important predisposing factor is the pre-existing lung diseases like tuberculosis, bronchiectasis, COPD, asthma and lung cancer, besides other risk factors which cause immunocompromised condition. Pulmonary mycosis may be caused by Aspergillus, Histoplasma, Cryptococcus or Pneumocystis, while pulmonary candidiasis is considerably rare. In patients with severe immunocompromised conditions, candidemia may develop as a serious condition which may lead to death.

To make the diagnosis of pulmonary mycosis, one should have strong suspicion towards the disease, based on the risk factors as described above and must be followed by laboratory tests, namely mycologic examination and, if possible, histopathologic examination. Diagnosis may be proven, possible or probable. The diagnosis is based on clinical features, risk factors and mycology and/or histology.

Treatment of pulmonary mycosis can be prophylactic, empiric, preemptive or causal. Causal treatment is given based on the proven existence of fungus either histologically or by culture, but is usually late. Empiric treatment is suggested in the strongly suspected patients even when mycologic test results are negative or not yet available, and preemptive treatment is based on positive result other than culture or histology.
For reatment of pulmonary mycosis amphotericin B, the azoles and echinocandins and TMP SMX are used, according to the severity, risk factors and of course the fungus as the causal or suspected agent. Amphotericin B is the most effective drug for all, but it is hardly used at present, due to the severe toxicity towards the kidneys that may lead to renal failure.

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CHRONIC THROMBOEMBOLIC PULMONARY HYPERTENSION – LATEST TREATMENTS

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ABSTRACT

Chronic thromboembolic pulmonary hypertension (CTEPH) is a form of pulmonary hypertension which results from incomplete resolution of acute pulmonary emboli, with formation of fibrotic material which obstructs large and distal pulmonary vasculature. The condition is largely underdiagnosed as early symptoms are subtle and mild. In advance cases, this condition results in right ventricular and respiratory failure.

Surgical Pulmonary Endarterectomy (PEA) is the mainstay of therapy which involves extensive luminal dissection of the chronic clot and fibrotic material of the entire pulmonary arterial tree. In experienced centres, perioperative mortality and mortality is low. Successful surgery restores normal hemodynamics and complete resolution of symptoms. The technique is discussed in this lecture.

Medical therapy is largely supportive with lifelong anticoagulation. Latest developments include pulmonary balloon angioplasty (PBA) and new drugs. Guanylate cyclase stimulator (Riociquat), phosphodiesterase5 inhibitors, endothelin receptor antagonists, prostanoids are several drugs that are used in this therapy. A short review of their action and effectiveness will be presented.

ANALGESIA CONSIDERATIONS FOR PATIENTS WITH RESPIRATORY PROBLEMS

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ABSTRACT

Alleviation and treatment of pain is a basic human right, especially in patients with co-existing disease, such as patients with respiratory problem. Chest pain which related to respiratory disease is common but underlying pathophysiology of respiratory chest pain are poorly understood.

The general principle for managing all pain is to initiate prompt, appropriate treatment to alleviate pain, ensuring a favorable benefit to adverse effect profile. The conceptual framework of the pain ladder, a stepwise approach, which originally described for patients with cancer-related pain, is now widely used for the management of all types of pain, including pain related to respiratory problems.

Optimal pain therapy is an essential requirement to maintain or to restore pulmonary function. Inadequate pain relief can result in increased stress response, sleep deprivation, disorientation, anxiety which in turn have potential to worsening underlying respiratory problems, and may be a risk factor of developing to chronic pain.

Adequate pain control may needs a combination of agents and techniques. Several routes of administration exist for pain relief. A balanced analgesic regimen consisting of regional analgesia with local anesthetics and NSAIDs should be used as first step. Intravenous opioid titration or patient controlled analgesia (PCA) with opioids, in combination with NSAIDs, might be appropriate for moderate to severe pain. Opioid titration will avoiding opioid-induced respiratory depression. Interventional pain procedure for respiratory pain is indicated when satisfactory analgesic control not achieved despite optimal pharmacologic management.

Keywords : pain management; respiratory disease;

Respiratory related pain

Chest pain which related respiratory disease is common but underlying pathophysiology of respiratory chest pain are poorly understood and studies of its pathophysiologi, clinical course, and management are limited.

Respiratory chest pain most commonly arises from parietal pleura (including thediaphragmatic pleura), chest wall, and the mediastinal structures.[1] The pleura costalis,or parietal pleura, lines the inner thoracic cavity, including the diaphragm andmediastinum, whereas the pleura pulmonalis, or visceral pleura, covers the entiresurface of the lung, including the interlobar fissures.[2] Although the two surfaces embryologically originate from the same coelomic membrane, their microscopicanatomy differs, with clinically important distinctions. The peripheral part of thediaphragm and costal portion of the parietal pleura are innervated by somaticintercostal nerves, thus pain felt in these areas is often localized to the cutaneousdistribution of the involved neurons over the adjacent chest wall. The centralportion of the diaphragm is innervated by the phrenic nerve, and central diaphragm irritation is referred to the ipsilateral shoulder tip or even the neck. The visceral pleura is extensively innervated by pulmonary branches of the vagus nerve and sympathetic

trunk, with no specific nociceptors.[1] Therefore, the presence of a localized pleuritic chest pain indicates involvement of the parietal pleura. Pains arising from the parietal pleura or chest wall are often exaggerated during deep respiration, coughing/sneezing, or body trunk movement involving the chestwall.

The intensity of pain may vary amongst patients with the same pathology, from asymptomatic to agonizing, and is not an indicator of the underlying cause. The description of the pain may also vary significantly amongst patients, for example, from sharp to dull, from burning to catching. The temporal evolution of the pain can be useful. Sudden onset of pain may accompany spontaneous pneumothorax or a rib fracture,whereas pain arising from malignant involvement of the pleura is often of insidious onset. Intercostal neuritis has been listed as a differential diagnosis of respiratory chest pain, but is not common.[3]

Parietal pleural inflammation is commonly termed pleurisy, a localized inflammation of the parietal pleura, which clinically produces a sharp localized pain, made worse ondeep inspiration or coughing, and occasionally twisting or bending movements. Apleural rub may be heard over the site of localized pleuritic pain. Although dry pleurisy occurs, pleural inflammation is generally associated with an exudative pleural effusion. Direct infiltration of the chest wall by a malignancy involving the parietal pleurafrequently produces a chronic dull ache localized to the relevant anatomic region,although referred neuropathic pain from intercostal nerve involvement is possible. Less frequently, trauma to the chest wall, ribs, or vertebrae may present in a similar way.

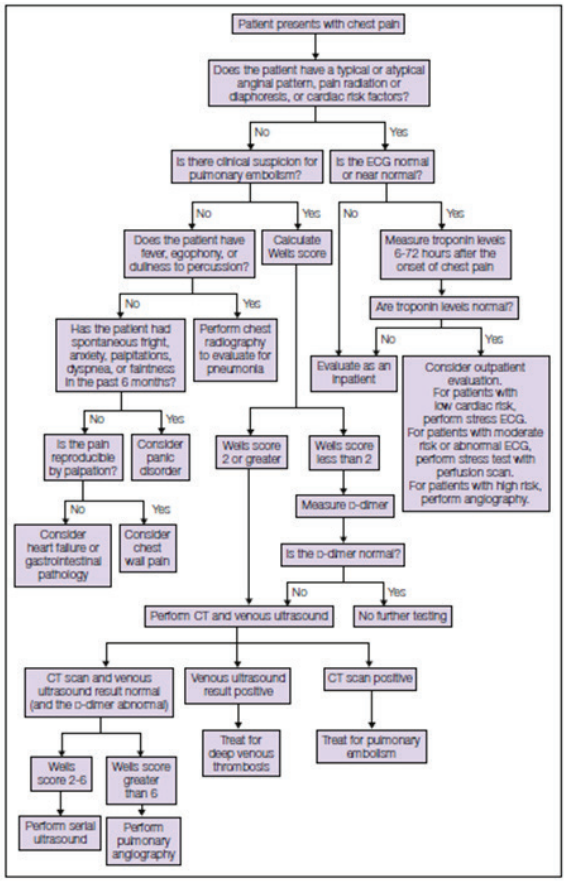

Figure 1. Algorithm for the diagnosisof chest pain in an outpatientsetting. The Wells score equalsthe sumof points for seven items: clinical signsand symptoms of deep venous thrombosis(DVT; minimum of leg swelling and painwith palpation of deep veins), 3 points;an alternative diagnosis is less likely thanpulmonary embolism (PE), 3 points; heartrate >100 beats/min, 1.5 points; immobilizationor surgery in the previous 4 weeks,1.5 points; previous DVT/PE, 1.5 points;hemoptysis, 1 point; malignancy (beingtreated, treated in the last 6 months, orpalliative), 1 point. CT, computed tomography;ECG, eletrocardiography. (FromCayley WE Jr 2005 Diagnosing the causeof chest pain. American Family Physician72:2012–2021. Copyright © 2005 AmericanAcademy of Family Physicians.)

Management Principles

The general principle for the control of all pain is to initiate prompt, appropriate treatment,at the correct dosage, ensuring a favorable benefit to adverse effect profile. TheWorld Health Organization introduced the conceptual framework of the pain ladder,guiding physicians to adopt a stepwise approach to the treatment of patients withpain.[4] Although originally described for patients with cancer-related pain, the concept is now widely used for the management of all types of pain. Optimal pain therapy is an essential requirement in the postoperative period to maintain or to restorepulmonary function. Several routes of administration exist for pain relief. Frequently, adequate paincontrol needs a combination of agents and techniques. A balanced analgesic regimen consisting ofregional analgesia with local anaesthetics andNSAIDs should be used. However, careful intravenous opioid titration or patient controlled analgesia(PCA) with opioids, in combination with NSAIDs,might be equally appropriate.

Agents

Opioids are highly effective for postoperativeanalgesia, although they carry a higher risk of respiratory depression, which is especially dangerous inpatients with COPD. Doses must be reduced by 50% of the standard adult dose in elderly patients with COPD in order to limit adverse events while maintaining an equivalent level of analgesia. PCA with opioids can be used systemically (intravenous administration) or with regional techniques (e.g.epidural administration) in elderly patients. Morphine is the most widely used and presently the mostsuitable drug for PCA in the elderly.[5] Close monitoring and evaluation of the patient throughout theperioperative period is required to ensure the appropriate and successful use of PCA. Pulse-oximetry isvery helpful for this purpose. Studies have indicated that, after acute pain control, PCA with morphine should be initiated at a dosage of 1 or 1.5mg perdose, with a lockout period of 5–7 minutes. Continuous background infusions of opioids are contraindicated.5 PCA is well-accepted by older patients, they attain comparable levels of analgesia and areequally satisfied with their pain control as younger patients.[6] However, when patients are unable to manage a PCA, the nurse or physician can titrate intravenous morphine. Intravenous morphine is titrated as a bolus of 2mg (bodyweight ≤60kg) or 3mg (bodyweight >60kg) in 5-minute intervals untilcomplete pain relief is achieved.[7]

Tramadol is a synthetic, weak opioid agonist and an inhibitor of monoamine neurotransmitterreuptake. Unlike other opioids, tramadol has noclinically relevant effects on respiratory or cardiovascular parameters at recommended doses.[8] Tramadol may prove particularly useful in patients withpoor cardiopulmonary function, including the elderly,the obese and COPD patients. In patients withimpairedhepatic or renal function, tramadol shouldbe used instead of NSAIDs.[8] The most common adverseevents (incidence of 1.6–6.1%) are nausea, dizziness,drowsiness, sweating, vomiting and dry mouth.[8] Recently, continuous intravenous tramadolhas been shown to be an alternative to neuraxialor systemic opioids for the management of post-thoracotomy pain.[9]

NSAIDs such as ibuprofen, naproxen, or diclofenacare effective postoperative analgesics. Unless theyare contraindicated or there is a strong concern abouthaemostasis or peptic ulceration, a scheduled parenteral, rectal or oral NSAID, should be added tothe patientspain regimen in order to reduce opioid consumption, enhance analgesia and decrease inflammatory mediators. Clinical benefits include less drowsiness and lack of respiratory adverse effects.This is important in patients with COPD where respiratory depression is especially dangerous.

Regional Techniques

Patient-controlled epidural analgesia (PCEA),using an opioid either alone or in combination with alocal anaesthetic, has been proven to be beneficial inthe management of pain relief after major surgery.[10] Seventy patients >70 years of age undergoing major abdominal surgery received either combined epidural analgesia and general anaesthesia followed by postoperative PCEA, using a mixture of 0.125% bupivacaine and 0.5µg/ml sufentanil, or general anaesthesia followed by PCA with intravenous morphine. Postoperatively, the group received the bupivacaine and sufentanil mixture via aPCEA pump programmed to deliver a 2 or 3ml boluswith a lockout interval of 12 minutes and a background infusion of 3–5 ml/h. The PCA group received an initial loading dose of intravenous morphine up to 5mg. Then the PCA pump was programmed to deliver a bolus of intravenous morphine 1.5mg with a lockout interval of 8 minutes. The epidural route using local anaesthetics and an opioidprovided better pain relief and improved mental status and bowel activity.[10]

Nerve Blocks

Intercostal nerve blocks using local anesthetic injection (eg, 0.25–0.5% bupivacaine or1%–2% lidocaine) provide reversible regional analgesia and are effective in controllingacute pain (eg, with rib fractures, or postthoracotomy) and chronic thoracic pain. Inpatients with pain of neuropathic origin, repeated blocks may afford permanent relief.Pneumothorax is a known but uncommon complication (1%). Paravertebral nerveblocks may be a useful adjunct in the treatment of postthoracotomy/thoracoscopypain, multiple rib fractures, and chronic pain syndromes.[11,12] Thoracic epidural with local anesthetic plus opioid, and intrathecal opioid analgesictechniques can provide effective pain control in postthoracotomy patients.

Non-Pharmacological Approaches

Chronic obstructive pulmonary disease (COPD) is a disease state characterized by aprogressive airflow limitation that is partly reversible after optimal pharmacologicaltreatment.[13] Although the pulmonary dysfunction may explain to a certain extentthe degree of severity of each day's symptoms of dyspnea and fatigue, it does notappear to be the foremost determining factor of exercisetolerance, survival,disease-specific quality of life, hospital readmission rate, and daily physicalactivity level in COPD patients.Pulmonary rehabilitation is a very important part in the management of patientswith moderate-to-severe COPD, which should probably be combined with acontinuum of self-management.[14] In fact, the World Health Organization advocatesadding pulmonary rehabilitation programs to the chronic disease management ofpatients with moderate-to-very severe COPD.

In general, exercise training in patients with COPD follows the principles of exercisetraining in the healthy elderly. Programs generally consist of a warm-up, a core program in which at least 30 minutes of active exercise is included, and a cooling down.Close supervision and proper monitoring ensure safety during the program.

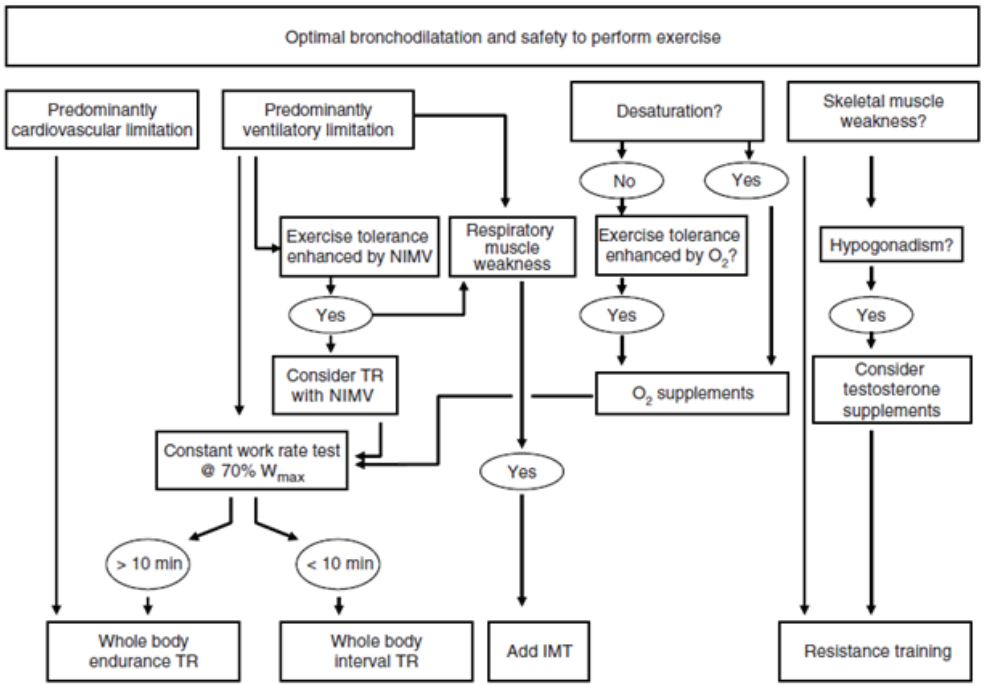

Figure 2 Empirical algorithm that could help the clinician to prescribe exercise therapy toindividual patients. Based on the exercise limitation of the patient (investigated in anincremental exercise test) and further clinical findings, different training strategies orcombinations can be prescribed. Typical cutoffs are: (i) respiratorymuscle weakness: PImax<60% predicted; (ii) hypogonadism: total serum testosterone: <400 ng/dL; and (iii)desaturation: saturation upon exercise <85%. Constant work rate test @ 70% Wmax is anexercise performed at 70% of the peak work rate from the incremental test. Abbreviations:Wmax, maximum work-rate duringprogressive cycle ergometry; PImax, maximum inspiratorypressure; TR, training; NIMV, non-invasive mechanical ventilation; IMT, inspiratory muscletraining.

Resistance Training

Whole-body endurance training (i.e., treadmill walking and ergometry cycling) has frequently been used to improve functional and peak exercise capacity in patients with COPD.[15] Nevertheless, a clear loss of fat-free mass and skeletal muscle dysfunction have been shown to be related to a decreased exercise capacity in COPD, irrespective of the degree of severity of the pulmonary impairment. It therefore seems reasonable to add resistance training to pulmonary rehabilitation of COPD. Resistance training is an exercise modality in which small muscle groups aretrained byrepetitive lifting (three series of eight repetitions) of relatively heavy weights(~75% of the one-repetition maximum = the maximum load that can be moved only once over the full range of motion without compensatory movements).

Resistance training has shown to be a very effective exercise intervention toimprove peak force of large muscle groups of the upper and lower extremities COPDpatients.[15] Indeed, changes in functional exercise capacity and disease-specificquality of life have been found to be similar in COPD patients following 12 weeks ofresistance or endurance training. Unfortunately, no studies have beenperformed to investigate

intramuscular changes following resistance training inCOPD patients. Then again, Jubrias and colleagues have recently shown large increasesin oxidative capacity (+57%) inhealthy elderly following six months of resistancetraining, accompanied by a rise in mitochondrialvolume density and quadricepsfemoris muscle size.[16]

The low demand on the respiratory system and, in turn, a lower sensation of dyspnea are most probably major advantages of resistance training compared withwhole-body endurance training. In fact, the moderate load on the impairedrespiratory system remained stable over time while the training intensity of there sistance training increased during pulmonary rehabilitation in patients withmoderate-to-severe COPD.[17]

Endurance Training

Endurance training has shown to be effective to improve disease-specific quality of lifeand endurance exercise capacity in patients with moderate-to-severe COPD, asreviewed by Lacasse and colleagues.18 Nevertheless, heterogeneous results havebeen found among patients withmoderate-to-severe COPD. Therefore,recent studies have focussed on enhancing the effects of endurance training in COPD by combining it with other treatment modalities, such as resistance training, respiratory muscle endurance training, inspiratory pressure support (IPS), long-acting bronchodilators,or supplemental oxygen.

Neuromuscular Electrical Stimulation

Unfortunately, active participation in pulmonary rehabilitation programs does not appear to be feasible in COPD patients with prolonged respiratory failure or with very severe daily complaints of dyspnea.[19] Therefore, other interventions should be considered to prepare these patients for active participation in an inpatient or outpatient pulmonary rehabilitation program. Neuromuscular electrical stimulation might be an interesting option. It can be used for skeletal muscle strengthening; maintenance of skeletal muscle mass and strength during prolonged periods of immobilization; and for selective muscle retraining, without pain, muscle lesions, or other adverse effects.[20] Indeed, in COPD patients with prolonged respiratory failure, it has shown toimprove skeletal muscle function and to reduce the number of days needed to make atransfer from bed to chair (from 14 to 11 days).[19] Moreover, neuromuscularelectrical stimulation has also shown to be a powerfulhome-based intervention toimprove skeletal muscle force, peak oxygen consumption, and symptoms of fatigue inCOPD patients with high baseline scores on the dyspnea scale of the Medical ResearchCouncil.[21] In fact, patients were able to continue the neuromuscular electricalstimulation at home during acute exacerbations of COPD and all patients were able tocomplete the intervention.[21] The positive effects of neuromuscular electricalstimulation appear to be limited to weak anddeconditioned COPD patients. Indeed, theeffects of six-week neuromuscular electrical stimulation of the quadriceps femorismuscles on skeletal muscle function and structure were rather modest in COPD patientswith well-preserved functional exercise capacity, skeletal muscle strength, and skeletalmuscle mass.

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MANAGEMENT OF ACUTE RESPIRATORY EMERGENCY CASES TSUNAMI BANTEN 2018

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ABSTRACT

Tsunami, Very large water waves generated by various types of disturbances on the ocean floor. This disorder can be in the form of earthquakes, plate shifts, or volcanic eruptions. Tsunami banten,December,23, 2018. The tsunami that occurred was caused by the eruption of Mount Krakatau.

Resulting in a death toll of 292 people, 3,976 people injured, 8 people missing and 33,136 people displaced (BNPN Pandeglang Banten)

Besides being injured, many Sunda Strait tsunami victims were also found to have aspiration pneumonia. This infection occurs because the victim drowns and makes foreign objects, including sea water, fill the lungs

Near drowning, It is still alive after the sinking event, and can cause serious secondary complications including death. Drowning, The sinking process begins with the damage to the respiratory system because the channel is under the surface of water (submersion) or water covers the face (immersion)

Near drowning occurs when the victim cannot breathe in water for a period of time. During sinking, oxygen intake will decrease and the body's main system can stop due to lack of oxygen. Pathogenesis, Near Drowning → Panik (Inspiration Effort reflex) air water aspiration and Asphyxia laryngospasm and Hypoxemia / Acidosis → Hypoxemia Airway occlusion by water & particles present in water → Aspiration pneumonia Aspiration pneumonia, complications of pulmonary aspiration.Lung aspiration is the entry of food, stomach acid, saliva, or other foreign objects into the lungs which can trigger lung infection,diagnosis :

- Blood tests include complete blood count, blood gas analysis, blood culture, electrolyte levels, and kidney function.
- Examination of sputum, for example sputum culture.
- Scan tests that include chest X-rays or CT scans. Bronchoscopy can also be performed, namely an examination procedure using a device equipped with a camera to see the throat down to the lower airway.

Bronchoscopy, Medical action that aims to visualize the trachea and bronchi, through a bronchoscope, which functions in diagnostic and therapeutic procedures for lung disease.
Case of tsunami victims :

1. Washing is done after a few days (3-7 days).
2. On the first day of the bronchial toilet the patient's condition improved.
3. Because of other complications and delay in initial treatment, patients who had bronchial toilet experienced worsening and died.

PULMONARY REHABILITATION OF RESPIRATORY DISEASE ON DISASTER

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ABSTRACT

Patient with respiratory disease has difficulty in gaining or maintaining optimal function of their respiratory quality, thus will get even worsen if involved as a disaster's victim or survival with disabling general condition.

Recent disasters such as earthquake, mount erruption, tsunami or flood, landslide, liquifraction or forrest fire, have brought to light the importance of disaster preparedness and planning for people with chronic diseases, especially related with respiratory disease. Lesson from Hurricane Katrina, more than 24% of hospital visits were due to exacerbations of chronic disease and evacuees with chronic disease were nearly twice as likely to present with acute symptoms on arrival to shelters.

People with chronic respiratory disease such as COPD are uniquely vulnerable to disasters, as many individuals with COPD are elderly, have physical impairments, and may be dependent on supplemental oxygen. It is currently unknown if people with COPD are engaging in general or disease specific disaster preparedness. It is important to assess the existing disaster preparedness of individuals with COPD, in order to establish if they are adequately prepared for disasters.

Post-disaster pulmonary rehabilitation program will enhance optimum pulmonary compliance and general endurance as tolerated to the survival victim with chronic respiratory disease and aid them coping their activity daily living.

Keyword: pulmonary rehabilitation, respiratory disease, natural disaster

Background

The disaster risks have been increasing recently due to the emergence of climate-related hazards which cause problems especially in coastal cities. Although the risks are obvious, many cities in Indonesia, which are surrounding by variety of potential natural disasters do not seem to have enough capacity to cope with the challenges and less disaster preparedness.[1,2]

The richness of natural resources along with the geographically located in the ring of fire mountain formations in equator region placed Indonesia as a supermarket of natural disaster. Pascapurnama et al studied some natural disasters in Indonesia to develop ideas on how to minimize health risks following natural disasters and to ensure good quality of life for people. For the past decade, Indonesia, a disaster-prone country, has been struck by natural disasters such as earthquake, volcano erruption, tsunami or flood, landslide, liquifraction or forrest fire, have brought to light the importance of disaster preparedness, mitigation of special needs people with certain kind of disease or disability and further emerging post disaster planning.[1,2,3]

Respiratory Disease related to Disaster

During disaster with many kind of causes, if related with some impact in respiratory disease, it should

became awareness because every living creatures are breathing, and this mechanism is disrupted in the disaster setting and it could be live threatening.[1,2]

Following such natural disasters, aside from the number of deaths and damaged infrastructure, the threat posed by health risks also looms, especially the emergence of infectious diseases. Some of following infectious diseases are likely to occur: diarrhea, also acute respiratory infections (ARI) which may interfere normal breathing and caused by virus or bacteria, dengue, malaria, measles, and tetanus. Natural disasters also result in "aftereffects" such as displaced populations (including internally displaced persons (IDPs) and refugees), poor sanitation, overcrowded space, and limited health supplies in evacuation center that might increase the possibility of infectious disease outbreak and worsen conditions for survivors. Aftereffects increase the transmission of infectious disease among survivors. The spread of a disease becomes more likely as the number of evacuated people increases. Aside from environmental changes and poor situations at evacuation centers, it is considered that people's knowledge and awareness of health risks also becomes one factor determining the occurrence of the infectious diseases. To tackle these and other challenges, collaboration for undertaking preventive measures of post-disaster infectious diseases should be integrated into disaster risk reduction (DRR) and management plans, and must be done not only by government, non-government organization (NGO) and non-profit organization (NPO), but also by public health and humanitarian professionals for the community.[2]

The direct cause of respiratory disturbance by many inhalant materials will enter the respiratory system during periods of disaster, developed inflammation of the airway, allergic reaction, obstructions, infections, irritations, suffocations might happened. The indirect cause of respiratory disturbance by the neuromuscular problem cause by traumatic brain injury, high spinal cord injury and multiple thoracic trauma that effect the breathing mechanism, impaired of lung compliance or central cough mechanism.[3]

People with chronic diseases, especially related with respiratory disease its self, such as chronic respiratory disease, in example; COPD determined to be worsen after a disaster. These disasters may exacerbate chronic respiratory diseases, such as chronic obstructive pulmonary disease (COPD). In the aftermath of the disaster, COPD patients often endure limited access to medication, medical equipment, and/or medical supplies. However, no systematic investigation has examined the impact of natural disasters on patients with COPD. [1,3,4]

Similar with an earthquake with tsunami that was happened in Aceh, Sumatera, Indonesia on the year of 2004, a magnitude 9.0 occurred in Japan on March 11, 2011, most severely affecting the Tohoku region on the northeast coast of the country. A devastating tsunami followed the earthquake and caused widespread damage on Japan's eastern coast. Approximately 20,000 people were killed or went missing, and over 380,000 houses were destroyed. In the aftermath of this catastrophe, we dealt with respiratory emergencies at a regional medical center set up in Ishinomaki to deal with the disaster's aftermath.[3,4]

Kobayashi et al identified 100 COPD patients (112 episodes) who presented at the emergency department and required hospitalization within 6 months after the Tohoku disaster. During the 6-month study period, 63 patients with exacerbations (68 episodes) presented at the emergency department requiring hospitalization. Five patients were hospitalized twice during the study period. The number of patients hospitalized due to COPD exacerbations each week is shown the total number increased 1.5- and 1.3-fold compared to the corresponding periods in 2010 and 2009, respectively. The number of patients increased during the period from 3 to 5 weeks after the earthquake and then decreased.

Kobayashi classified the patients into 3 groups in terms of the time of hospitalization after the disaster: the acute phase (first 2 weeks after the earthquake), the sub acute phase (from weeks 3 to 5), and the chronic phase (from 6 weeks to 6 months). The number of patients admitted while in the sub acute phase significantly increased compared to the corresponding periods in 2010 and 2009. There were no significant differences between the patients in the acute plus sub acute phase group as compared to those in the chronic phase group in terms of age, FEV1, percentage of predicted FEV1, regular medication, or long-term oxygen therapy. However, the deterioration in ADL upon admission was significantly different between the groups. The ADLs of patients were significantly decreased compared to that before the earthquake in the acute phase and sub acute phase group. In contrast, no reduction in ADL was observed during the chronic phase. All patients with exacerbations were treated in accordance with the consensus guidelines. Mechanical ventilation was required in 7 patients with NPPV and 1 patient with invasive mechanical ventilation. The in-hospital and 90-day mortalities of patients with exacerbations of COPD were 5.9% (4/68) and 13.6% (8/59), respectively. At 90 days, 9 patients had been lost to follow-up. Six patients were treated conservatively, and 2 patients underwent thoracoscopic surgeries. Seven patients were cured, but 1 patient with very severe COPD required invasive mechanical ventilation and died due to complicating pneumonia. Six and 7 patients with pneumothorax were identified in the years 2009 and 2010, respectively. No patient included in the study experienced a pulmonary embolism.[4]

The findings of our study indicate that the Great East Japan Earthquake had a strong negative impact on clinical out- comes among COPD patients. In the acute phase of the disaster, patients with very severe COPD sought refuge in our hospital and were provided with oxygen therapy. A population 3 times as big was admitted due to exacerbated symptoms. During the chronic phase, the frequency of admission due to exacerbations returned to baseline levels. During the acute phase, most of the COPD patients who presented at the hospital were seeking oxygen therapy. In Japan, home oxygen therapy is widely used for patients with chronic respiratory failure and is covered by the national healthcare insurance system. Since the Hanshin–Awaji earthquake in 1995, medical personnel and oxygen-service providers have recognized the importance of managing oxygen-dependent patients during a disaster and have established emergency operation measures. The wide-scale disaster of 2011, however, was more catastrophic than predicted in even the most pessimistic scenarios. Fortunately, we were able to accept many oxygen-dependent patients who were normally treated at other clinics in addition to our own outpatients.[4]

The evacuees were not hospitalized because casualties who were seriously ill occupied all of the hospital beds. Instead, each COPD patient was provided with a continuous oxygen supply via the central gas piping system in the outpatient ward. The unexpectedly high number of patients exceeded the facility's capacity for sound care. Therefore, on March 14, we established a temporary oxygen therapy center inside the hospital using electric oxygen concentrators. This area had been used as a rehabilitation center before the disaster and lacked an oxygen piping system. Trained nurses in the Respiratory Medicine Department were assigned to the evacuation center to provide medical care, and respiratory physicians visited each outpatient every day. Nevertheless, there was a high incidence of symptom exacerbation among those patients staying at the evacuation center. Symptom severity was likely exacerbated by the facility's poor insulation and the interruption of regular treatment. Some patients had been drenched by the tsunami, while others, prior to their hospital visit, stayed in houses or shelters that not only lacked oxygen supplies but also lacked heating systems or water supplies. Some patients were also deprived of their prescribed drugs, and this interruption of regular treatment may have partly contributed to the worsening of symptoms.[4]

The symptoms of many patients worsened during the sub acute phase. The number of patients hospitalized due to exacerbated symptoms was 3 times higher than those hospitalized during the corresponding period in 2009 or 2010. First, interruption of regular treatment may have resulted in increase in exacerbations of COPD. In addition to the factors cited above, tracheobronchial infections may be associated with worsened COPD symptoms. Previous reports demonstrated that respiratory infections increased in the aftermath of a massive earthquake. In Ishinomaki and the surrounding areas, habitants suffered insufficient fuel supplies, power failures, water and food shortages, and an inability to maintain the appropriate level of personal hygiene. Cold winter temperatures and damaged houses or emergency shelters compounded these conditions. Such unfavorable conditions are likely to result in the increased occurrence of respiratory infections. The inhalation of dust and fine particles from rubble and tsunami- sludge also make breathing difficult. It has been reported that air pollution is an important risk factor for the exacerbation of COPD. Many buildings in Ishinomaki were destroyed by the tsunami, and a thick layer of mud covered the entire area. Thus, chemicals, particulates, and biological materials from debris and tsunami-sludge may have contributed to the worsening of respiratory symptoms among COPD patients in the area hit by the tsunami. The deterioration of ADLs in the acute and sub acute phases after the disaster resulted in increased number of hospitalizations. It was previously reported that physical disability was an independent risk factor for death after the Hanshin–Awaji earthquake and the 1999 Taiwan earthquake. However, those reports investigated mortality in the acute phase, but not hospitalizations in the sub acute or chronic phases.[4]

Recent reports have also demonstrated that physical inactivity is a risk factor for symptom aggravation and mortality in COPD. After the earthquake and tsunami in the Ishinomaki region, over 40,000 evacuees stayed at crowded emergency shelters, where they had to lie down on the floor without beds. Elderly patients with COPD were largely sedentary, which led to ADL deterioration. Furthermore, impaired ADLs and poor oral hygiene induced swallowing dysfunction, which can in turn exacerbate COPD. The number of patients presenting with COPD exacerbations declined in the chronic phase as compared to the sub acute phase. The recovery of water and food supplies, the restoration of vital infrastructure and medical services, and the improvement in living conditions may have contributed to this phenomenon. In Ishinomaki and the surrounding cities, medical relief teams circulated around community evacuation centers and prescribed medications for patients with chronic disease. These efforts minimized the interruption of treatment during the chronic phase. Diagnoses of pneumothorax and pulmonary embolism should be considered in COPD patients reporting exacerbated symptoms, even in the aftermath of natural disasters. In this study, we identified 8 patients with pneumothorax, and none with pulmonary embolism. Patients whose symptoms had worsened underwent comprehensive evaluation, including chest radiography, which led to the detection of pneumothorax in several patients.[4]

This Kobayashi report is the first retrospective study to determine outcomes in patients with COPD who experienced a large-scale natural disaster in a developed nation with an aging population. Although pulmonary complications, such as chest trauma or respiratory infection, are commonly recognized after natural disasters, the impact on the outcomes of COPD patients had not previously been clarified. The results of our study indicate that patients with COPD will suffer substantially in the aftermath of natural disasters.

Pulmonary Rehabilitation on Post-Disaster Setting

Since medical rehabilitation services are often poorly developed in disaster affected regions and not highly prioritized by responding teams, physical and rehabilitation medicine (PRM) has historically been underemphasized in global disaster planning and response. Recent development of the specialties

of "disaster medicine" and "disaster rehabilitation" has raised awareness of the critical importance of rehabilitation intervention during the immediate post-disaster emergency response.[3]

The Committee of Rehabilitation on Disaster Relief (CRDR) of International Society of Physical and Rehabilitation Medicine (ISPRM) seeks to 'effectively focus the resources of ISPRM and its membership on optimizing the health, functioning and quality of life of persons who sustain injuries or impairments due to a large-scale natural disaster". As defined by the WHO's International Classification of Functioning, Disability, and Health (ICF), "functioning" comprises health domains such as body functions and structures as well as activity and participation in the persons' environment. [1,3]

Strategic goals developed by the CRDR to achieve its mission include providing post-disaster rehabilitative services, including an emergency rapid response team capability; facilitating coordination between various disaster responders, including humanitarian relief and governmental organizations, disabled persons organizations and local providers over the disaster response, providing rehabilitation education and training to disaster responders, host personnel, caregiver and community.

Specifically for survival victims with previous chronic respiratory disease, such as COPD or the aftermath respiratory patient to respiratory disruption due to disaster, based on functional management of rehabilitation, to be concerned into these criteria, consist of neuromuscular impairment (restrictive problems), obstructive problems, airway secretion problems, endurance problem and ADL limitation. [1,3,5]

Pulmonary rehabilitation has been defined as "a multidimensional continuum of services directed to persons with pulmonary disease and their families, usually by an interdisciplinary team of specialists, with the goal of achieving and maintaining the individual's maximum level of independence and functioning in the community". [5]

Pulmonary Rehabilitation interventions can include exercise, respiratory muscle rest and support, education, emotional support, oxygen, airway secretion clearance, promoting compliance with medical care, facilitating return to work, and a more active and emotionally satisfying life. These goals are appropriate for any patients with diminished respiratory reserve whether due to obstructive or intrinsic pulmonary diseases (oxygenation impairment) or neuromuscular weakness (ventilatory impairment). 5

Regarding the Kobayashi report on dividing the post disaster as acute, sub acute and chronic setting, in the first and applicable management in every kind of setting is breathing retraining. Effective breathing management, combine with relaxation techniques, pulsed lip breathing, diaphragm breathing and energy conservation in activity daily living as tolerated with the situation.[4,5]

Airway secretion clearance is crucial because exacerbations of COPD are common in the disaster setting and frequently caused by trapping of airway secretions in the peripheral airways. The patient's cough may be weak or ineffective as a result of increased airway collapse in more central airways, and frequent bouts of coughing are fatiguing. Providing a comprehensive airway secretion management in rehabilitation intervention is also one of the very important strategies to apply.[3,5]

The first main goals in Pulmonary Rehabilitation in Disaster Setting are maintaining the pulmonary compliance, lung growth in developmental lung or children, and chest-wall mobility, also continuously

maintain normal alveolar ventilation by assisting inspiratory muscles as needed and provide functional coughs by assisting expiratory muscles.[3,5]

Pulmonary compliance is lost because the ability to expand the lungs to the predicted inspiratory capacity is lost as the VC decreases. As the VC decreases, the largest breath that one can take can only expand a small portion of the lungs. Like limb articulations and other soft tissues, regular range of motion (ROM) is required to prevent chest-wall contractures and lung restriction. This can only be achieved by providing deep insufflations, air stacking, or nocturnal NIV. This is a function of bulbar muscle integrity. Patients who cannot close the glottis and, therefore, cannot air stack, must be passively insufflated using a Cough-Assist (Respironics International Inc., Murrysville, PA) or pressure-cycling ventilator at pressures of 40 to 70 cm H2O. The maximum passive insufflation volume can be termed the "Lung Insufflation Capacity" or LIC. 5 Adaptation to ADL in further setting depends on patient conditions and regular endurance exercise such sit to stand, walking and functional ADL exercise can be applied as tolerated.[5]

Conclusion

The large-scale natural disasters have a negative impact on clinical outcomes among chronic respiratory disease such as COPD patients as survival victims in the affected area. Further studies are required to determine how various types of natural disasters influence clinical outcomes. [4]

Post disaster rehabilitation results in faster medical recovery, fewer complications, and greater improvement of functional outcomes. Improved outcomes, in turn, contribute to greater social integration and community participation, which help build post disaster society (as opposed to expending limited health and social services). [3]

Many reports result suggest that respiratory physicians, in cooperation with disaster specialists, along with rehabilitation medicine team should develop strategies and preparedness for the comprehensive management of COPD and other respiratory post disaster-related patients in the aftermath of natural disasters. [3,4]

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SATURDAY, 27th JULY 2019

RADIOLOGIC FINDINGS IN NEONATAL RESPIRATORY DISTRES

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ABSTRACT

Respiratory distress in the neonates is an emergency problem, affecting up to 7% of all term newborns, and is increasingly common in prematurity.

Respiratory distress is one of the most common reasons an infant is admitted to the neonatal intensive care unit. Fifteen percent of term infants and 29% of late preterm infants admitted to the neonatal intensive care unit develop significant respiratory morbidity; this is even higher for infants born before 34 weeks' gestation. Certain risk factors increase the likelihood of neonatal respiratory disease. These factors include prematurity, caesarian section delivery, gestational diabetes, maternal chorioamnionitis, such as oligohydramnios or structural lung abnormalities. However, predicting which infants will become symptomatic is not always possible before birth. Regardless of the cause, if not recognized and managed quickly, respiratory distress can escalate to respiratory failure and cardiopulmonary arrest.

Therefore, it is important to recognize the signs and symptoms of respiratory distress, interpretating the radiograph chest finding, differentiate various causes before determining initiate management strategies to prevent significant complications or death.

Failure to readily recognize symptoms, diagnosis and treat underlying cause of respiratory distress in the newborn can lead to short- and long-term complications, including chronic lung disease, respiratory failure, and even death

Definition, Signs, Symptoms
Respiratory distress in the newborn is recognized as one or more signs of increased work of breathing, such as tachypnea, nasal flaring, chest retractions, or grunting. Normally, the newborn's respiratory rate is 30 to 60 breaths per minute. Tachypnea is defined as a respiratory rate greater than 60 breaths per minute Tachypnea is a compensatory mechanism for hypercarbia, hypoxemia, or acidosis (both metabolic and respiratory) making it a common but nonspecific finding in a large variety of respiratory, cardiovascular, metabolic, or systemic diseases.

The causes of respiratory distress in a newborn are diverse and multisystemic. Respiratory disease can be divided into surgical or non surgical. Respiratory distress may result from developmental abnormalities that occur before or after birth. Early developmental malformations include tracheoesophageal fistula, bronchopulmonary sequestration (abnormal mass of pulmonary tissue not connected to the tracheobronchial tree), and bronchogenic cysts (abnormal branching of the tracheobronchial tree). Later in gestation, parenchymal lung malformations, including congenital cystic adenomatoid malformation or pulmonary hypoplasia from congenital diaphragmatic hernia or severe oligohydramnios, may develop. More common respiratory diseases, such as TTN, RDS, neonatal pneumonia, MAS, and persistent pulmonary hypertension of the newborn (PPHN), result from complications during the prenatal to postnatal transition period.

Respiratory Distress Syndrome (HMD)
RDS, also known as hyaline membrane disease, is a common cause of respiratory disease in the premature

infant. RDS is also seen in infants whose mothers have diabetes in pregnancy. RDS is caused by a deficiency of alveolar surfactant, which increases surface tension in alveoli, resulting in microatelectasis and low lung volumes. Surfactant deficiency appears as diffuse fine granular infiltrates on radiograph.

Infants with RDS typically present within the first several hours of life, often immediately after delivery. Initial chest X ray findings are low volumes and diffuse reticular granular opacities, air bronchogram and poor lung expansion. Cardiac size is normal.

Most common cause of death in live born infants. More common in infants of diabetic mothers.
Acute complication such as alveolar rupture with pneumothorax, pneumomediastinum,pulmonary interstisiel pneumonia.

Treatment : prenatal prevention with treatment of mother, maternal steroid will cross placenta and increase surfactant Surfactan administration, mechanical ventilation (PEEP) .

Meconium Aspiration Syndrome
MAS occurs when the fetus passes meconium before birth. Infants born through MSAF are at risk for aspiration of meconium in utero or immediately after birth. Any infant who is born through MSAF and develops respiratory distress after delivery, which cannot be attributed to another cause, is diagnosed as having MAS.

The typical chest radiograph initially appears streaky with diffuse parenchymal infiltrates. In time, lungs become hyperinflated with patchy areas of atelectasis and infiltrate amid alveolar distension. Surfactant is inactivated by the bile acids in meconium, resulting in localized atelectasis, so alternatively, radiographs may resemble those of RDS with low lung volumes.

Common complications of meconium aspiration syndrome include pneumothorax (left upper) and persistent pulmonary hypertension of the newborn (right upper) characterized by cyanosis with normal lung fields and decreased pulmonary vascular markings.

Transient tachypnoea of the newborn (TTN) is one of the commonest causes of respiratory distress in newborns especially in late-preterm and term infants.[1] TTN results from delay in clearance of fetal alveolar fluid after birth. The management consists of supportive care, with symptoms generally resolving by 24-72 hours of age.[2]

Pathophysiology
Fetal lung fluid is essential for normal lung development and is secreted by lung epithelium. A few days prior to the onset of labour, lung fluid production decreases. During labour, maternal epinephrine and glucocorticoids stimulate absorption of alveolar fluid through activation of an amiloride-sensitive epithelial sodium channel.[8,9] "Vaginal squeeze" only accounts for a fraction of the fluid absorption.

Newborns especially in late-preterm and term infants.1TTN results from delay in clearance of fetal alveolar fluid after birth. The management consists of supportive care, with symptoms generally resolving by 24-72 hours of age.[2]

TTN results from disturbance in the mechanisms responsible for fetal lung fluid clearance. Risk factors

for TTN includeelective caesarean section, delivery before completing 39 weeks of gestation, maternal diabetes, maternal asthma, male gender, and small or large-for-gestational age.[4-7]

Chest X-ray (AP and Lateral): Prominent ill-defined central markings suggestive of vascular engorgement radiating out from hilum, prominence of interlobar fissures (fluid), small pleural effusions, mild hyperinflation, enlarged cardiac silhouette. Chest X-ray may be delayed for 4-6 hours as a proportion of the infants with provisional diagnosis of TTN would improve in 4-6 hours10and may not require chest X-ray.

NEONATAL PNEUMONIA AND PERSISTENT PULMONARY HYPERTENSION OF THE NEWBORN (PPHN)
Neonatal Pulmonary inferction can occur in utero, during passage through the birth canal or just after birth
Predisposing factors : prolonged rupture of the mambranes, maternal vaginal infection, placental infection, contamination of infant with maternal faecal material or fetal sepsis.

The chest X ray : a diffuse infiltrate, may be reticular pattern or patchy opacities , can be unilateral or bilateral

PPHN
A consequence of pneumonia, hypoxia, birth asphyxia and sepsis, and is associated with pulmonary hypoplasia.

It may also develop without any obvious predisposing.
The Chest X Ray : evidence of pneumonia, but the lung fields may have minimal changes or there may be oligaemia

BRONCHOPULMONARY DYSPLASIA (BPD)
A Combination of barotraumas, prolonged ventilation support, infection, deficiency of anti-oxidant defences and altered inflammatory response in the preterm lung.

The Chest X Ray :
Lungs are hyperinflated , ill defined , corarse , reticular opacities interstisiel interspersed with small cystic like areas.

This progress over weeks to month : larger cystic, become bubbly lungs , and may have atelectasis and fibrosis

RESPIRATORY DISTRESS NEEDS INTERVENTION/SURGICAL
CONGENITAL LOBAR EMPHYSEMA (CLE)
Progressive overdistention of pulmonary lobe due to bronchial obstruction with ball valve phenomenon
Air enters involved region but has difficulty leaving

Radiography findings : unilateral radiodens lobe that becomes progressively hyperlucent and hyperexpanded.

CONGENITAL PULMONARY AIRWAY MALFORMATION (CPAM)
Heterogeneous group of cystic and noncystic lung lesion resulting from early airway maldevelopment.
Imaging findings : multicystic mass with variable amount of air/fluid in cysts. By imaging categorize into large cyts, small cysts, microcystic/solid types. Lesion very often of mixed type

CONGENITAL DIAPHRAGMATIC HERNIA

Herniation of abdominal contents into chest via defect in diaphragm, most commonly posterior on left (Bochdalek).

Imaging findings on chest X ray : Bubbly , bowel like lucencies in chest. Left more common than right (5:1). May contain variable abdominal contents : stomach, small and large bowel, liver.

NEONATAL PNEUMOTHORAX

Presence of gas in pleural cavity between visceral and parietal pleura. Tension pneumothorax is large PTX with contralateral mediastinal shift.

Imaging findings : Chest X-Ray : hyperlucent lung with contralateral mediastinal shift. Cross table lateral or left lateral decubitus may be helpful to make diagnosis.

Treatment : supportive care if patient is clinically stable. Needle evacuation may be used without chest tube for mild symptom, Needle evacuation also used for urgent evacuation of a PTX in acute setting.

PULMONARY SEQUESTRATION

Congenital area of abnormal lung that does not connect to bronchial tree or pulmonary arteries.
Imaging findings : lower lobe opacities that persist overtime. Left lower lobe > right lower lobe. Systemic arterial supplay. Occasionally occurs below the diaphragm.

Treatment : surgical resection in symptomatic cases

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LABORATORY PARAMETERS & METHOD OF CHOICE FOR HEALTH PROBLEM AFTER NATURAL DISASTER

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ABSTRACT

1. Introduction

Indonesia is one of the most disaster-prone countries in the world, regularly experiencing earthquakes, tsunamis, landslides, volcanic eruptions, flooding, and drought. Beside natural disaster, a human made disaster were currently more frequent, such as boom blast, chemical disaster, traffic accident, riots, etc. Disaster was not only resulting in number of deaths, injured people and the damaged health facilities, but it was also creating public health problems, for example the disaster related diseases, the broken water supply and sanitation facilities, electricity and psychologically traumatic issues among the victims.[1,2]

Laboratory testing is needed not in acute emergency phase. The role of laboratory after acute phase is; to identify the risk of infectious disease to become an outbreak.[3] In the other hand, non-communicable diseases such as cardiac and kidney diseases also need special attention.[4] The problem is, to perform laboratory testing require electricity, clean water, refrigerator, waste disposal etc. Laboratory should have a specific standard operating procedure, the method of choice and the laboratory parameters of choice should be performed in disaster situation.[3]

2. Disaster in Indonesia

Indonesia experienced in almost all type of natural disaster and human made disaster as well (Figure 1).

Figure 1. Disaster threat in Indonesia Adopted from (Triwahyu, 2019)2

In the year of 2014, USAID'S Office of U.S. Foreign Disaster Assistance (USAID/OFDA) published a recorded disaster in Indonesia from 1990 until 20141 as follow (Figure 2).

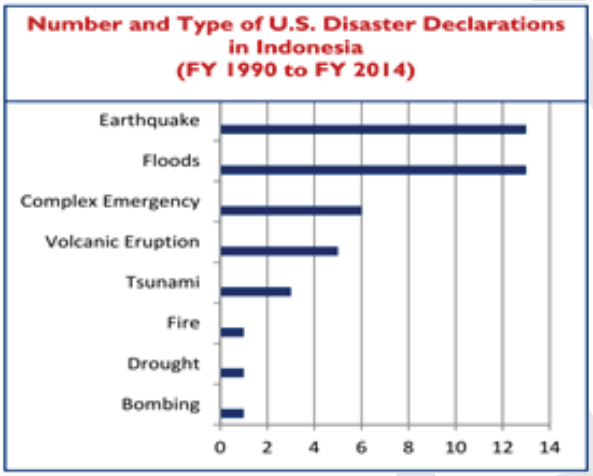

Figure 2. Number and type of Disaster in Indonesia from 1990 to 2014.
USAID'S Office of U.S. Foreign Disaster Assistance (USAID/OFDA)1 USOFF OF U.S. FOREIGN DISASTER ASSISTANCE (USAID/OFDA) USAID'S OFFICE OF U.S. FOREIGN DISASTER ASSISTANCE (USAID/OFDA)

3. Definition

Indonesia Disaster Definition:

(UU. NO 24 – 2007 ttg PENANGGULANGAN BENCANA)

Events or series of events threatening and disturbing life and community livelihood caused by nature and/or non-nature or human factors resulted in human casualties, environmental damage, loss of property and psychological impact.[5]

WHO Disaster Definition:

Any event that results in a precipitous or gradual decline in the overall health status of a community with which it is unable to cope adequately.[3]

4. Diseases-related disaster

Disaster-related diseases are often acute in nature, with the most serious requiring immediate triage to emergency and critical care facilities. The disaster emergency-critical care continuum begins with the identification of at-risk patients, followed by patient stabilization, and ultimately transfer to an alternate care facility or mobile hospital for comprehensive critical care. Gaps at the interfaces foreach of these settings leads to excess mortality and morbidity.[6] Injuries and deaths during or shortly after natural disasters are directly associated with fractures, lacerations, blunt trauma, crush injuries, projectile injuries, burn injuries and drowning.[7] Response to emergencies has traditionally focused on management of acute conditions such as trauma and infectious illnesses. However, noncommunicable diseases (NCDs) such as diabetes, hypertension, cardiovascular disease, cancer, and chronic lung disease are now leading causes of disability and death in low-income and middle-income countries (LMICs) and disaster-prone areas.[4]

4.1 Infectious Diseases-related Disaster

Increases in infectious disease transmission and outbreaks following natural disasters are associated with prolonged after-effects of the disaster. These after effects include displaced populations (internally displaced persons and refugees), environmental changes, increasing vector breeding sites, high exposure to and proliferation of disease vectors (rodents, mosquitoes), unplanned and overcrowded shelters, poor

water and sanitation conditions, poor nutritional status and poor personal hygiene, low levels of immunity to vaccine-preventable diseases or insufficient vaccination coverage, and limited access to healthcare services.[7]

4.1.1 Three clinical phases of natural disasters

In a review article done by Kouadio et al, from 21 publication, three clinical phases of natural disasters7 are summarized below;

a. Phase 1, the impact phase (lasting 0–4 days), is the period usually when victims are extricated and initial treatment of disaster-related injuries are provided.
b. Phase 2, the postimpact phase (4 days to 4 weeks), is the period when the first waves of infectious diseases (air-borne, food-borne and/or water-borne infections) might emerge.
c. Phase 3, recovery phase (after 4 weeks), is the period when symptoms of victims who have contracted infections with long incubation periods or those with latent-type infections may become clinically apparent. During this period, infectious diseases that are already endemic in the area as well as newly imported ones among the affected community may result in an epidemic. The breakdown of natural disasters recorded from 2000 to 2011 and potential secondarily-associated infectious diseases. (Table 1)

Table 1. Breakdown of natural disasters recorded from 2000 to 2011 and potential secondarily-associated infectious diseases

Country	Disaster event	Year(s)	Infectious disease outbreak following natural disaster
USA	Tornado	2011	Cutaneous mucormycosis
Japan	Earthquake	2011	Diarrhea (norovirus), influenza
Haiti	Earthquake	2010	Cholera
Cote d'Ivoire	Flood	2010	Dengue
Brazil	Flood	2008	Dengue
USA	Hurricane (Katrina)	2005	Diarrhea, TB
Pakistan	Earthquake	2005	Diarrhea, hepatitis E, ARI, measles, meningitis, tetanus
Dominican Republic	Flood	2004	Malaria
Bangladesh	Flood	2004	Diarrhea
Indonesia	Tsunami	2004	Diarrhea, hepatitis A and E, ARI, measles, meningitis, tetanus
Thailand	Tsunami	2004	Diarrhea
Iran	Earthquake (Bam)	2003	Diarrhea, ARI
Indonesia	Flood	2001–2003	Diarrhea
USA	Hurricane (Allison)	2001	Diarrhea
Taiwan	Typhoon (Nali)	2001	Leptospirosis
China	Typhoon (Nali)	2001	Leptospirosis
El Salvador	Earthquake	2001	Diarrhea, ARI
Thailand	Flood	2000	Leptospirosis
Mozambique	Flood	2000	Diarrhea
India (Mumbai)	Flood	2000	Leptospirosis

(Kouadio, 2012)[7]

Infectious disease outbreak following natural disaster shown in Table 1, are associated with prolonged after-effects of the disaster. The risk factors of those communicable diseases are figure out in Table 2.

Table 2. Risk factors and onset of communicable diseases following natural disasters

Major risk factors following natural disasters	Water-borne diseases			Air-borne/droplet diseases				Vector-borne diseases		Contamination from wounded injuries		Clinical phase of natural disasters		
	Diarrhea (cholera; dysentery)	*Leptospirosis*	*Hepatitis*	*ARI (pneumonia/ influenza)*	*Measles*	*Meningococcal meningitis*	*TB*	*Malaria*	*Dengue fever*	*Tetanus*	*Cutaneous mucormycosis*	*Impact phase (0–4 days)*	*Postimpact phase (4 days- 4 weeks)*	*Recovery phase (>4 weeks)*
Population displacement from nonendemic to endemic areas								✓	✓					✓
Overcrowding (close and multiple contacts)	✓			✓	✓	✓	✓						✓	
Stagnant water after flood and heavy rains	✓	✓						✓	✓					✓
Insufficient/contaminated water and poor sanitation conditions	✓		✓										✓	
High exposure and proliferation to disease vectors		✓						✓	✓					
Insufficient nutrient intake/ malnutrition	✓			✓	✓		✓							✓
Low vaccination coverage					✓									
Injuries										✓	✓		✓	✓

[1]Disasters do not carry diseases/epidemics. Disease risk factors need to be in place and exacerbated as a result of the after effects of the disaster.
ARI: Acute respiratory infection.

(Kouadio, 2012)[7]

4.2. Non communicable Disease following disaster

Disruption of access to healthcare facility in disaster made people with non communicable disease are at greatest risk of their condition worsening or even death. Non communicable disease include cancer, cardiovascular conditions, diabetes, renal diseases and respiratory diseases.[3] Disaster victims are at risk for acute myocardial infarctions, acute kidney injury (AKI), and sepsis. A conceptual framework impact of disasters on NCDs is presented in Figure 3.[8]

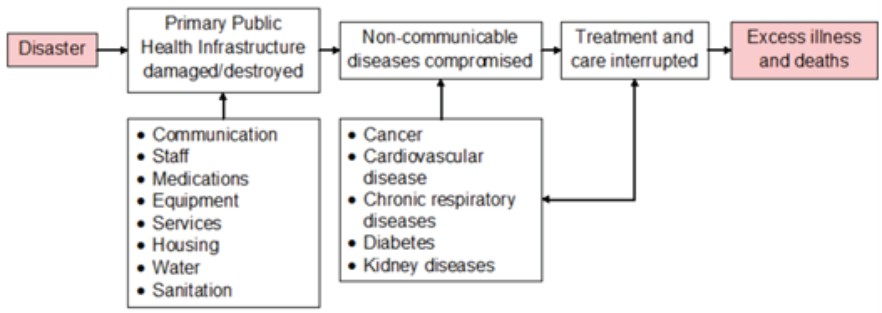

Figure 3. Conceptual framework impact of disasters on NCDs[8]

6. What is the Role of Laboratory in disaster situation?

6.1 Disaster Situation Analysis

Laboratory testing is not a priority in acute emergency phase. During the early stages of a disaster, laboratory services may be more orientated towards the identification of the major health problems rather than of the diagnosis of individual patients. Polluted water and poor sanitation, crowding, inadequate shelter and social disorganization are risk factors common to disasters and therefore likely to cause outbreaks of communicable diseases, which will require screening, diagnostic and other laboratory services. The clinical laboratory in the field should have a good coordination with the reference clinical/hospital laboratory for referral or for confirmatory tests.[9]

6.2 Infrastructure needed for laboratory establishment

The minimal requirement to establish temporary laboratory are[3,9,] at least;

- Electricity for minimal equipment
- Refrigerator for blood bank
- Microscope
- Clean water
- Waste disposal
- Personal protective equipment (PPE)

6.3 Laboratory Method of Choice

Survey done by Brock et al (2010)10 showed that respondents (in disaster) preferred patient-side testing (point-of-care testing) in the field over testing inside a vehicle or tent. This finding was strengthen by Kost's survey[12,13], that rapid diagnostic test (RDT) with point-of-care (POC) devices used in disasters should address the needs of first responders, who give high priority to contamination-free whole-blood sampling, superior performance pathogen detection, and HIV-1/2 blood donor screening. The use of POC testing accelerates triage, but more importantly, facilitates evidence-based practices necessary for the disaster-emergency critical care continuum. Point-of-care testing serves to harmonize the interface between disaster, emergency, and critical care. However, laboratory personel must pay attention to operating temperature range, storage conditions, sensitivity, specificity of the tests.

6.4. Laboratory Parameters needed in Disaster Situation

Based on the finding what is the majority of diseases found in disaster situation, the laboratory parameters should be the priority in disaster[13] is shown in figure 4.

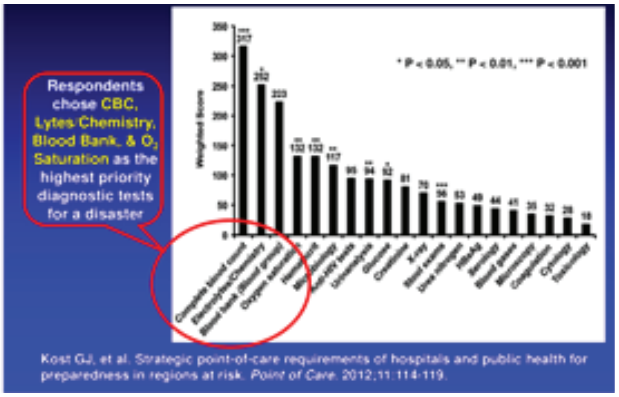

Figure 4. Respondents laboratory parameters choice as a priority in disaster.[13]

Brock et al[10] proposed the disaster Point of Care as shown in Figure 5.

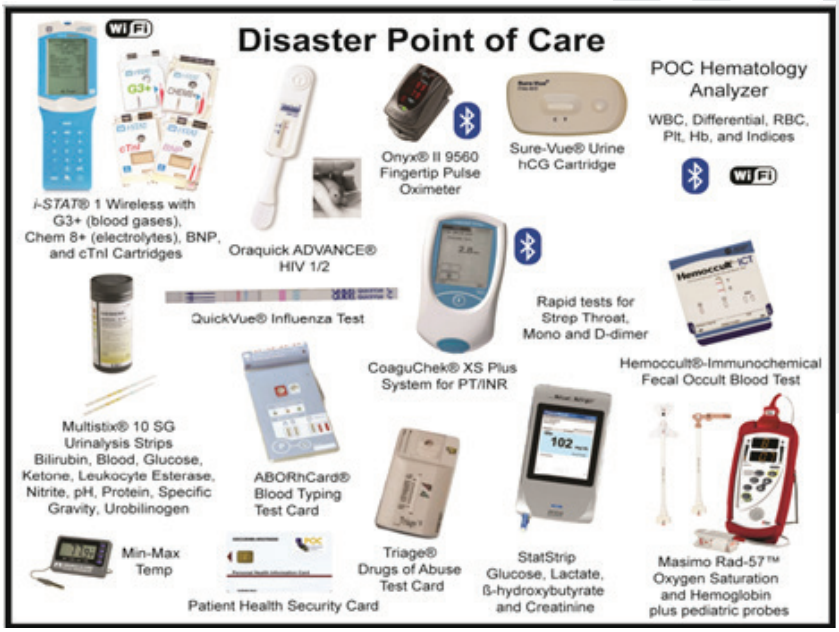

Figure 5. Disaster point of care
Brock KT. Disaster Point-of-Care: Non-Invasive Monitoring. YouTube Presentation.
Available at: http://www.youtube.com/watch?v=9ELSR7z0U4w.[10]

Multiplex molecular pathogen detection at the point of care helps clinicians and first responders to rapidly identify the microorganism causing sepsis and prompts early initiation of appropriate antimicrobial therapy. Respondents preferred single patient multiple pathogen testing in disaster situation [12] is shown in Figure 6.

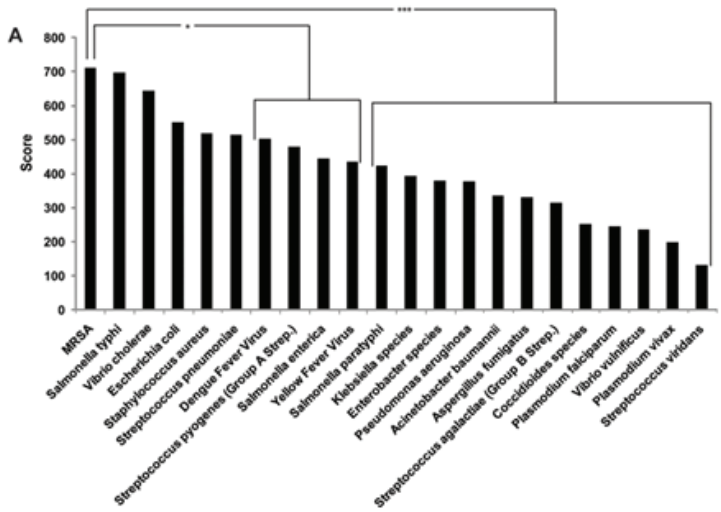

Figure 6. Respondents preferred single patient multiple pathogen testing in disaster situation

7. Conclusion

Clinical laboratory service in disaster situation is challenging regarding to disruption of electricity, water supply etc. The method of choice is rapid diagnostic test or point of care testing. However, quality control should always be performed and sensitivity specificity should be put into consideration. The laboratory parameter tested is depend on the first situation analysis or epidemiology, concern should be for both infectious disease and non-communicable disease.

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PAIN MANAGEMENT IN CRITICALLY ILL PATIENT

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ABSTRACT

Introduction
On March 11, 2011, a giant tsunami hit the East Japan facing the Pacific Ocean caused by a 9.0 Richter earthquake. The disaster killed more than twenty thousand people and crushed more thousands of buildings and natural environment. The energy was ranked 4th in energy level among all of the earthquake aroung the world since 1900. Another tsunami disaster happened in west of northern Sumatra on December 26, 2004. The tsunami affected at least five million people and killed 280,000 people.[1]

The main cause of death due to these disasters is drowning in the tsunami wave rather than being trapped under the building after the earthquake. Even for the victims who got rescued, they developed serious lung damage.1 Severe lung damage and pulmonary infections by the victims was caused by aspiration of sea water that contains sand, microorganisms, and heavy oil chemicals. In overcrowded camps, measles and acute respiratory infection had to be watched since it is transmitted easily. These conditions are described as "Tsunami lung"[2]

Respiratory Problem in tsunami victim
Drowning and near-drowning are an unexpected catastrophic events that occur in individuals who are often healthy and young. Drowning can cause great problem to all of the major organ system, with pulmonary and neurological damage as the main cause of morbidity and mortality. The abnormalities that can affect drowned individuals include renal failure and cardiovascular instability caused by ischemia, disseminated intravascular coagulation, and metabolic abnormalities.[3,4] Another complication that can cause death is infection caused by bacteria of fungi, with pneumonia as one of the most devastating infection.

Drowning can be divided into freswater drowning and seawater drowning. These two types of drowning are different because the seawater contains salt so that it has bigger osmotic pressure than freshwater. However, recent studies suggest that there is small difference between these two because the water volume that enters the lungs rarely causes electrolyte abnormality in a living person.[5,6] The lung disorders caused by drowning can be distinguished with segmental pneumonia or lobar pneumonia because it affects the entire lung since the water enters the lungs during respirations movements.[1]

Drowning caused by tsunami waves is different from drowning in normal seawater. This is due to various objects (sand, building debris, vehicles, chemicals, soil, wastes, oil, etc) that is carried away by the seawater that hit the land. As the results, the water aspirated by the victims contains various substances that causing more damaged to the lungs.[1]

Recent studies suggest that oil is the most damaging substances that causes the development of tsunami lung. This oil comes from gasoline, households, and heavy oil from factories and carried away by the tsunami wave to cover the sea surface. The drowning victims of the tsunami will attempt to go to the surface to breath. At this moment, they are very likely to aspirate these various oils and develops severe chemical-induced pneumonia. The dirty seawater that contains bacteria and more substances can cause infective

pneumonia that aggravates the conditions. In short, tsunami lung may be described as combination of bacterial pneumonia and mechanical or chemical induced pneumonia that affects the entire lungs.[1,7]

Chief pathogens of the pneumonia are: *(1) Gram-negative rods: Burkholderia pseudomallei, Chromobacterium violaceum, and Pseudomonas aeruginosa, (2) Gram-positive rods: Streptococcus pneumoniae, and (3) fungi: Pseudallescheria boydii.*8 In some cases of 2004 Indian Ocean tsunami, several types of infections caused by Aspergillus were reported.[9,10] However there are no *aspergillosis* cases were reported so far. Bacteria that often isolated from pneumonia caused by freshwater drowning include *Burkholderia psudomallei* and *Aeromonas,* while in seawater drowning are usually *Francisella philomiragia* or *Pseudomonas aeruginosa.* Common organisms that cause drowning-associated pneumonia are listed on table 1.[8]

Pathogen	Freshwater	Salt water	CFR (%)	Time to symptoms
Aeromonas spp.	+++	+	63.6	< 24 hours
Burkholderia pseudomallei	++		83.3	14 days
Chromobacterium violaceum	++		0.0	> 1 month
Francisella philomiragia	?	++	20.0	5 days
Klebsiella pneumoniae		+	0.0	4 days
Legionella spp.	+		66.7	4 days-6 weeks
Neisseria mucosa		+	100.0	< 24 hours
Pseudomonas aeruginosa	+	?	50.0	5 days
Shewanella putrefaciens		+	0.0	4 days
Vibrio spp.	?	+	100.0	4 days
Streptococcus pneumoniae	++	+	100.0	< 24 hours
Staphylococcus aureus	?	?	?	
Aspergillus spp.	?	+	0.0	7 days
Pseudallescheria boydii	?	?	80.0	> 1 month

Table 1. Type of environmental exposure related to organisms causing near-drowning-associated pneumonia8

a. Tsunami Lung

The common features in tsunami lung victims include adult respiratory distress syndrome (ARDS) caused by aspiration of sea water, chemicals, and other particles in early stage of a severe case. After 2 to 3 days of drowning, patient developed pneumonia caused by aspirated bacteria from tsunami wave. In some cases, pneumonia caused by tsunami lung was refractory to antibiotics. And after 2 or 3 weeks later after drowning, patient might developed mycotic infection as an abcess in lung and brain.[2]

a. Patient Case: Two-year-old Girl

Figure 1. Two-year-old girl's chest X-ray showed right upper lobe consolidation and infiltrative shadow in left upper lobe

This patient was a victim of 2011 Japan tsunami and transported to a hospital the following day after tsunami. Initially, she was suspected aspiration pneumonia and administered with ceftaxim. Therapy is switched to piperacillin because sputum culture showed a Pseudomonas aeruginosa infection, but the chest shadow worsened (Figure 1). Then, another test is conducted and a urinary antigen test for Legionella pneumophila was positive and her antibody titre rose to 1024-fold. Then, levofloxacin was administered and the shadow on the right superior lobe was improved.[11]

Even though her symptoms was improving, sudden tonic convulsions and conjugate deviation of both lower leg developed on day 23. A CT Scan of the brain showed a subarachnoid haemorrhage and hydrocephalus aroung the brainstem (figure 2). The brainstem haemorrhage are thought to be caused by a mycotic aneurysm, and the patient died on day 36.[11]

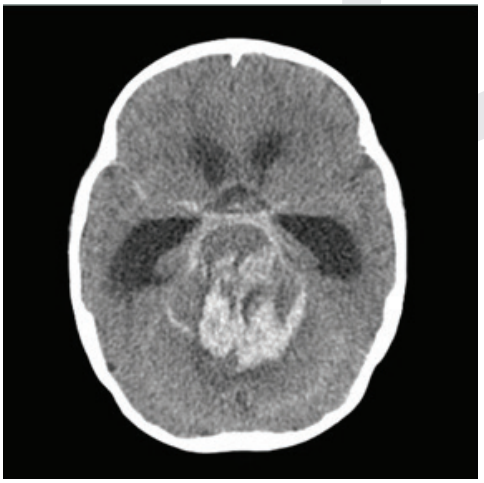

Figure 2. Computed tomography scan of the brain after convulsion shows bleeding around brainstem associated with subarachnoid haemorrhage and hydrocephalus[11]

b. Acute Respiratory Infection

After a disaster, communicable disease, especially those that affected the lung, is common in the victims population. Poor availability of sanitation facilities and clean water, population displacement, and inadequate health-care services were thought to be the main cause for the spread of diseases. These are often added by low vaccination status and poor underlying health of the affected population. The victims usually live in the overcrowded emergency shelters with limited daily living support and may facilitate the spreading of communicable diseases.[12]

Large caseload for respiratory infection in tsunami victims occured in the 1st month following the tsunami, nearly 90% of the total annual caseload. Four months after tsunami in aceh, there are 8,854 cases recorded compared to 10,029 annual case in 2003. Generous health services availability and active case finding may play role in this increase, but it is generally agreed that there are increases in acute respiratory infection after a tsunami. Interestingly, large proportion of the cases happened in adults. Only less than a third of the cases occurred in children under five. No direct explanation is available, but it could be caused by higher survival rates of adults in tsunami events.[13]

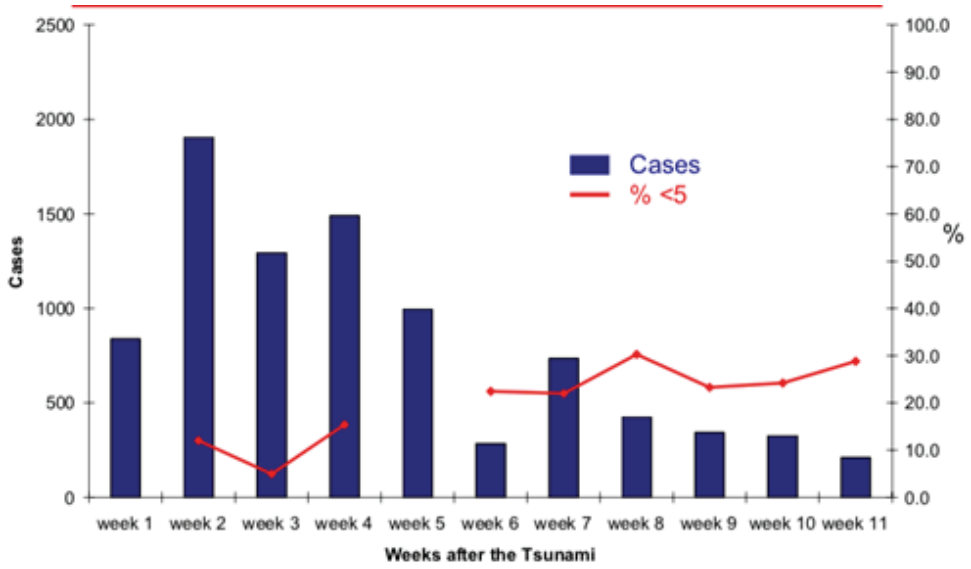

Fig 3.: Cases of pneumonia and % among children < 5: Aceh[13]

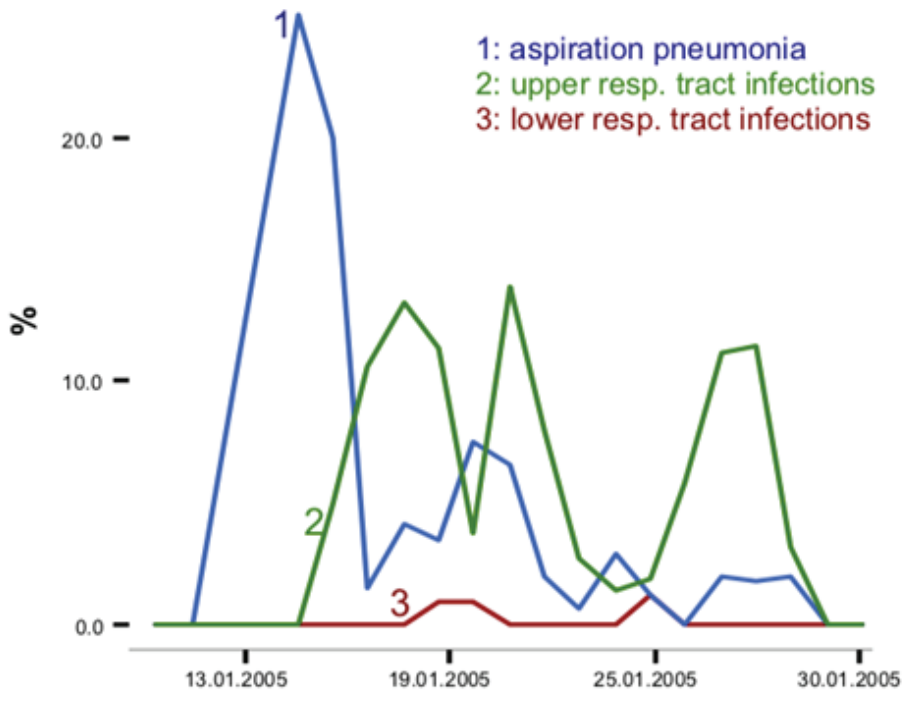

Fig 4. Respiratory tract infections ICRC hospital Banda Aceh: Jan 11-31[13]

c. Influenza

Influenza infection are observed having widespread morbidity during the pandemic of a H1N1 novel influenza in 2009. Epidemics of influenza infection after a natural disasters are not documented properly, however, after a few weeks of the Great Eastern Japan Earthquake in 2011, there are some cases reported in some evacuation centers.[14]

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PAIN MANAGEMENT IN CRITICALLY ILL PATIENT

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ABSTRACT

Introduction

Pain is a common and distressing symptom of patients in intensive care unit (ICU) and represents a major clinical, social, and economic problem. It has been reported that almost 80% of patients experience different intensities of pain during they are in ICU and identify it as a one of the greatest sources of stress. Such pain is problematic because produces adverse psychological and physiological response that includes increased heart rate, blood pressure, respiratory rate, neuroendocrine secretion and psychological distress. Failure to relieve pain produces a prolonged stress state, which can result in harmful multisystem effects and impair a patients recovery and discharge. The primary goal of acute pain management in ICU patients are pain control and attenuation of the negative physiologic and psychological consequences of unrelieved pain. Although, a number of recent surveys, reported that enhanced pain management was associated with improved patient outcome in the ICU and despite of pain research, guideline development, numerous awareness campaigns and intense educational efforts, pain remains currently under evaluated and undertreated in patients who are critically ill. Therefore, the importance of quality pain management in the ICU is inherently compelling and highly challenging.

Etiology and pain assessment in ICU patients

Although, adequate pain control is a basic human right, a number of factors complicate the management of pain in the critically ill patient. In particular, critically ill patients may experience pain due to their underlying disease or surgery, but also it may be result of various and painful medical procedures (procedural pain) such as inserting urinary catheter, nasogastric tube, chest tubes, tracheal suctioning, invasive lines, (arterial and central venous catheter) suture removal and routine nursing care. Nursing care procedures such as bathing, massage of back and pressure points, sheets change and repositioning are the most common painful procedures in ICU patients. Vazquez M et al. analyzing pain intensity during 330 turnings in 96 medical surgical patients and reported significantly increased pain score between rest and turning. The bolus of analgesic was used in less than 15% of the turnings. Further, although some ICU patients may be able to communicate, many critically ill patients with cognitive or communication problems due to stroke or brain injury, dementia, confusion, mechanical ventilation and concomitant use of sedatives, may have difficulty in reporting pain. Presence of the some causes mentioned above increases the likelihood for poor pain management, and worsens a patient's experience of pain. The first step in providing adequate pain relief for ICU patients is appropriate assessment.

Pain should be assessed by self-reporting scales in patients able to communicate, or by behavioral pain scores in patients unable to communicate. Even though various self report pain scales and behavioral pain scales specifically developed for use in critically ill adults are available, these are not always routinely used in the ICU. Patients' self reporting of their pain is the gold standard of pain assessment and provides the most valid measurement of pain.

The most widely used pain intensity scales are the Numeric Rating Scale (NRS) and Visual Analogue Scale (VAS) while Behavioral Pain Scale (BPS) is considered to be an alternative tool for assessing pain in critically ill, sedated, and mechanically ventilated patients. The BPS assesses pain through evaluation of facial expression, upper limb movements, and compliance with mechanical ventilation. A similar behavioral scale called the Critical Care Pain Observation Tool (CPOT) may also be used.
Types of Pain experienced by ICU patients

There are many causes that can lead to the development of pain in patients at risk in critically ill patients. These include trauma, surgical procedures, malignancies, burns, and other causes. Therefore, a proper classification of pain is needed to help establish sufficient pain management plans. Generally, we classify pain into the following groups:

1. Pain that is continuous and associated with surgical procedures.
2. Ongoing disease pain that is acute.
3. Pain that is intermittent and can be associated with procedures in the intensive care unit.
4. Pain that is chronic and had been present before the admission of the patients to the intensive care unit.

Other consequences of intensive care unit pain are the resulting compromises in oral cavity suction, bronchial tree suction, changing patients positions, wound care, removal of drain, insertion of catheters, and establishing IV accesses. Some long-term consequences can result from this pain, most importantly, the prolonged resulting pain that will significantly decrease patients' quality of life after they are discharged from the intensive care unit. All these types of pain are significantly related to sex, age, and prior pain conception with previous interventions. Despite being that common, pain is properly managed only in about 25% of intensive care unit patients.

Route of administration

The route of medication administration is an important consideration for the pharmacologic management of pain in the ICU setting. Intravenous administration is more commonly the route of choice in critically ill patients because of altered GI tract function that could lead to unpredictable absorption of medication.
The choice of intermittent vs. continuous infusion IV administration depends on factors such as the frequency and severity of pain, and the pharmacokinetics of the pain medication. the administration in bolus is associated with the variation in the peak plasma concentration, since the infusion maintains a more stable concentration, but can lead to accumulation of medication especially in patients with renal or liver failure.

Patient-controlled analgesia (PCA) is an effective method for administering analgesic medication and gives patients a sense of control over their pain, specially in postoperative settings. Patients can determine when and how much medication they receive, regardless of analgesic technique. However, this technique requires fully conscious and orientated patients which make use of PCA limited in ICU patients.

Intravenous administration is generally preferred over subcutaneous or intramuscular routes given potentially inadequate absorption due to regional hypoperfusion (e.g., shock, subcutaneous edema). Regional or neuraxial (spinal or epidural) modalities may also be used in ICU following selected patients and selected surgical procedures. Epidural analgesia (EA) is probably the most often used regional anaesthetic technique in the ICU. EA should be proposed in critically ill patients, such as post-operative other thoracic, abdominal surgery, major vascular surgery and orthopedic surgery or trauma patients, typically.

The major disadvantages of epidural analgesia are the rare but catastrophic complications such as infection, epidural hematoma formation and nerve damage, which can occurred in ICU patients who have a high risk of developing these complications.

Pharmacotherapy

In January 2013, the Society of Critical Care Medicine (SCCM) published the Clinical Practice Guidelines for the Management of Pain, Agitation, and Delirium in Adult Patients in the Intensive Care Unit. Table 1. shows the commonly used pain management drugs and recommended doses. there are no data to support the preference of analgesic over the other.

Opioids

Opioids are the primary medications for managing pain in critically ill patients because of potency, concomitant mild sedative and anxiolytic properties, and their ability to be administered by multiple routes. Recommended opioids include fentanyl, remifentanil, morphine, and hydromorphone. The choice of opioid and the dosing regimen should be individualized based on drug's potency, pharmacokinetics and pharmacodynamic profiles, side effect profile, patient comorbidities, and function of specific organ systems, in particular the liver and kidneys.

Morphine sulfate is the most frequently used opioid in the ICU and has been traditionally a first-line opioid for the treatment of severe pain. Morphine has a half-life of 1.5 to 2h after intravenous administration in normal subjects, but in ICU patient, distribution volume and protein binding may be abnormal. Therefore, the patients can respond very differently to morphine doses in terms of the analgesic effect but also in terms of the side effects. Although, morphine, such as all opioids, may lead to respiratory depression, it is noteworthy to point out that the morphine-6-glucuronide metabolite is more potent than morphine itself, and that accumulation can occur, especially in patients with renal impairment. Therefore, morphine use should be avoided for patients with known renal insufficiency or failure. Side effects include histamine release, sedation, nausea, ileus, constipation, and spasm of the sphincter of Oddi. Morphine sulfate should be administered intravenously and titrated to effect. The loading dose of 0.05 mg/kg (2–5 mg) should be given over 5 to 15 min. In most patients, the average maintenance dose is 4 to 6 mg/h and should be administered at the dosing interval of 1–2 hrs. Continuous IV morphine can be administered with an initial 2–5 mg bolus dose followed by 1 mg/h.

Fentanyl is synthetic opioid roughly 100 times the potency of morphine, which does not cause histamine release and was preferred analgesic agent for critically ill patients with hemodynamic instability. It efficacy is due to its lipid solubility (600 times more lipid soluble than morphine), so if used > 4 hours fentanyl must be used in the lowest tolerated dose to prevent prolonged effects. With prolonged infusion, the half-life increases dramatically from 30 – 60 minutes to 9–16 h and care must be taken to adjust infusion rate with time.

Fentanyl causes only minor hemodynamic changes and does not affect cardiac inotropic. The dose range for fentanyl infusions is variable and some patients may re-quire higher doses. Bolus dose of 25–100 µg, with subsequent doses of 0.25–0.5 µg/kg every 15–30 minutes might be a good first alternative to morphine in treating acute painful conditions. Alternatively, a bolus dose of 1–2 mcg/kg (25–100 mcg) may be administered followed by initiation of the continuous infusion. Most patients will be adequately treated with 1 to 2 µg/kg/hr (25–200 µg/h infusion).

Remifentanil is a fast-acting drug and presents an equally fast recovery. It is 150 –200 times more potent than morphine. Its metabolism does not depend on the liver. Analgesia-based sedation with remifentanil is a useful option for mechanically ventilated patients and it can be used in patients that need frequent neurological assessment. Studies have shown a shorter duration of mechanical ventilation and quicker ICU discharge with remifentanil compared with other opioids. It offers precise control of analgesia for painful procedures in ICU patients and has a highly predictable onset and offset, with a stable context sensitive half-time (3–10 min). No need for initial dose adjustment is required for patients with impaired renal and hepatic function. Therefore, analgesia-based sedation with remifentanil has been introduced as an option in ICU patients. Remifentanil can be administrated in higher doses than are normally used with other opioids without concerns about accumulation and the possibility of unpredictable and/or delayed recovery. Most ICU patients can be managed without bolus doses; if required, a bolus of 0.5–1 μg/kg is usually sufficient. It is recommended that remifentanil infusions should be started at 6–9 μg/kg/h and then titrated in the range dose 0.5–15 μg/kg/h. Some authors recommended dose to 60 μg/kg/h. Even under these controlled conditions, this practice has not found widespread use be- cause of the associated incidence of hypotension and bradycardia.

Hydromorphone is a semisynthetic opioid agonist that, like fentanyl, has a more rapid onset of analgesia (within 30 minutes) and a short half-life (2–4 hours). While the duration of action is similar to morphine, it does not stimulate histamine release. Hydromorphone is primarily metabolized in the liver to an active metabolite hidromorphone-3-glucuronide, but it is not clinically significant. Hydromorphone is potent respiratory depressant and may accumulate in patients with renal failure, resulting in neuroexcitation and cognitive impairment. Dosing begins at 0.2 to 0.6 mg and titrated by 0.5 mg increments. Most patients requiring 1 to 2 mg every 1 to 2 hrs. In addition, if given as an intravenous continuous infusion the dose should be 0, 5 –3 mg/h.

Tramadol is a centrally acting opioid-like drug, and acts by binding to the μ opiate receptor where it is a pure agonist like morphine and inhibits adrenaline and serotonin reuptake. It is used to treat moderate to severe pain. The most common adverse effect is typical to other opioids and includes nausea, vomiting, dizziness drowsiness, dry mouth and headache. However, tramadol produces less respiratory and cardiovascular depression than morphine, and euphoria and constipation are also less common.
Recommended dosage of 100 mg can be administered as an initial bolus. During the 90 minutes following the initial bolus further doses of 50 mg may be given every 30 minutes, up to a total dose of 250 mg including the initial bolus. Subsequent doses should be 50 mg or 100 mg 4 to 6 hourly up to a total daily dose of 400 mg.

Non-Opioid Analgesics

Non-opioid analgesics are indicated for use in management of mild to moderate pain and moderate to severe pain with adjunctive opioid analgesics. Potential advantages of multimodal analgesia, that involves combination of analgesics with different mechanisms of action, include improved analgesia, effective analgesia with lower opioid doses, and decreased risk of opioid-related adverse effects.
Non-steroidal anti-inflammatory drugs (NSAIDs) have opioid sparing effect but this has not been sufficiently investigated in ICU patients. Although the use of NSAIDs is still controversial, they may be used as adjuncts to opioid therapy. The most common side effect include gastro-intestinal bleeding, renal dysfunction and inhibition of platelet function.

All parenteral NSAIDs should be avoided in patients with preexisting renal insufficiency, asthma, hypoperfusion, advanced age, concomitant use of steroids and anti- coagulants, situations that are frequently observed in ICU patients. Treatment should be limited to the mini-mum dosage for the shortest possible time, not to exceed five days.

Acetaminophen (paracetamol) was approved for intravenous use in 2010 and is commonly administered for the short-term treatment of mild to moderate pain and febrile critically ill patients with infection. It differs from the available opioids and NSAIDs, since paracetamol does not increase incidence of nausea, vomiting, and respiratory depression that can occur with opioids, or the platelet dysfunction, gastritis, and renal toxicity that are associated with NSAIDs. Although, represents a relatively good safety profile, there is limited information regarding IV use in critically ill patients. Research to date has described that paracetamol can cause transient abnormalities of liver function and may cause hypotension in critically ill patients. Acute liver failure is the most serious potential complication of the use of paracetamol. The key criteria for assessing potential hepatotoxicity with conventional doses of paracetamol may include hypoxic injury, altered pharmacokinetics, relative over-dosage, muscle glutathione depletion, malnutrition, dehydration, older age and alcoholism which is often seen in critically ill patients. The British National Formulary (BNF) suggests to administer a maximum daily infusion dose of 3 g in adults in these patient groups.

Randomized, placebo-controlled trial that investigating the safety and efficacy of paracetamol in febrile ICU patients with known or suspected infection is currently underway study (the HEAT - Permissive Hyperthermia trough Avoidance of Paracetamol in Known or Suspected Infection in the Intensive Care Unit) (18). The results of this trial are expected to publish in early 2015 and should provide essential information on efficacy and safety of paracetamol in febrile critically ill patients. The recommended dose for IV acetaminophen is 1 g every 6 h with a maximum allowable dose of 4 g/daily.

Lidocaine is commonly used for regional anesthesia and nerve blocks, however, recent clinical studies demonstrated that intravenous perioperative administration of lidocaine can lead to better postoperative analgesia, reduced opioid consumption, improved intestinal motility and decrease hospital length of stay. Although, the analgesic effect depends on dose, there is considerable individual variability in pharmacokinetic response to lidocaine infusions. Serum steady state is achieved following a bolus of 1.5–2.0 mg/kg of lidocaine and infusion rates of 0.9–3.6 mg/kg/h. these doses generally result in plasma levels of 1.3–3.7 µg/ml, which provide a small margin of safety. Large doses have better analgesic effect but induce systemic lidocaine's toxicity. Lidocaine induces analgesia when serum ranges are kept at 1–5 µg/m. Although the half-life of the drug is only 120 minutes, the analgesia provided by systemic lidocaine is prolonged, over days or even weeks. With regard to analgesia, it has been reported that intravenous lidocaine produces three different pain relief stages: the first is during infusion and 30 to 60 minutes after its end; the second is a transient stage approximately 6h after infusion; and the third stage appears 24 to 48h after infusion and continues for 21 to 47 days. Intravenous lidocaine should not be used in patients with arrhythmias, heart failure, coronary artery disease, Adams-Stokes, or heart blocks. Caution should be taken also when using lidocaine in patients with hepatic or renal failure, sinus bradycardia, and incomplete branch block since possible accumulation of lidocaine or its metabolites may lead to toxic phenomena.

Although there is no clear consensus on the dosage regimen, many studies have used a bolus dose of 100 mg or 1.5 mg/kg at least half an hour before surgical incision, followed by an infusion of 1.33–3 mg/kg/h intra-operatively and continued after operation variably up to 24 h. the application of continuous lidocaine

has not been documented in the ICU in controlled studies and more study is needed to confirm beneficial effects of lidocaine in critically ill patients.

Regional anesthesia and analgesia

Regional anesthesia and analgesia although, not commonly used as a primary modality for analgesia in critically ill patients, can help to improve respiratory and bowel function, mental status and patient comfort secondary to its opioid-sparing effects. It minimizes patient discomfort and reduces the physiological and psychological stress, as in non-critical patients. Limitations for the use of regional anesthetic techniques are mainly associated with bleeding risks, coagulation disorders, hemodynamic disturbances and difficulties in neurologic assessment. the use of regional analgesia in the ICU settings should evaluate the risk and benefits due to limited cooperation of the patient, and the indication for it use should be carefully assessed regarding to patients clinical condition.

As mentioned above, epidural analgesia is probably the regional anaesthetic technique most often used in the ICU, but nerve blocks and other sophisticated techniques started in the operating room may also be used for pain relief in critically ill patients and should not be discontinued when the patient is transferred to ICU.

Table 1
Commonly used pain management drugs and recommended doses.

Drug	Elimination Half-Life	Peak Effect (IV)	Suggested Dosage	Comments
Morphine	2-4 h	30 min	2-5 mg bolus 1-10 mg/h infusion	Avoid in hemodynamically unstable patients. Active metabolite accumulates in renal dysfunction. May cause itching due to histamine release.
Fentanyl	2-5 h	4 min	25-100 µg bolus 25-200 µg/h infusion	Fastest onset and shortest duration. Accumulation with hepatic impairment. Muscle rigidity.
Remifentanil	3-10 min	1-3 min	0.5-1 mcg/kg IV bolus 0.5-15 µg/kg/h infusion	No accumulation in hepatic/renal failure. Use IBW if body weight >130% IBW
Hydro- morphone	2-4 h	20 min	0.5-2 mg bolus and 0.2 to 0.6 mg every 1-2 h intermittent 0.5-3 mg/h infusion	fterapeutic option in patients tolerant to morphine/ fentanyl. Accumulation with hepatic/renal impairment 5-10x more potent than morphine.

Tramadol	5-6 h	45 min	100 mg bolus and 50 mg every 30 min up to 250 mg including the initial bolus. total daily dose of 400 mg.	fte elimination of tramadol may be prolonged in hepatic/renal impairment Contraindicated in patients on MAOI or epilepsy.
Acetamino- phen	2-3 h	15 min	1 g every 6 h	May cause hypotension when given by infusion and may cause liver and kidney damage, when taken at higher than recommended doses (overdose).
Lidocaine	1,5-2 h	45-90 s	100 mg or 1.5–2 mg/kg at least half an hour before surgical incision, followed by an infusion of 1.33–3 mg/kg/h intraoperatively	Avoid in patients with arrhythmias, heart failure, coronary artery disease, Adams-Stokes, or heart blocks. Caution should be taken in patients with hepatic or renal failure, sinus bradycardia and incomplete branch block.

Conclusion

Pain management is an essential component of quality care delivery for the critically ill patient. The patients in ICU often suffer from undertreated and unrecognized pain, with potentially serious physical and psychological effects. The availability of a wide range of treatment options together with the recognized importance of adequate management enables better understand, evaluate and manage pain in the critically ill patient. It's therefore important for clinicians to recognize a patient's pain profile and rational choice of pain medication should be based upon individual needs and desired effect of analgesic. Effective pain management is a moral imperative and

THE STRENGTH OF MULTIDISCIPLINE IN DISASTER EMERGENCY RESPONSE

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ABSTRACT

Disasters are unexpected events, since it is unknown when, where, and how they will occur. They create chaos, risk of injury or illness and loss of life or property. When disasters occur, there is often a mismatch between resources and needs, magnifying the chaos, risks and losses

Since the last 10 years the data show that the incidence of disaster increased significantly. Indonesia is a vulnerable area for disaster are located on three tectonic plate, namely Autralia Plate, the Pasific plate and Eurasia plate, causing high potential for earthquakes, volcanic eruptions and landslides.
Indonesia is geographically located in the ring of fire that runs along the Pasific plate which is the most active plate in the word. It's contributes the largest almost 90% earthquake events on earth and almost all of them are the major earthquakes in the world.

In the face of increasing disaster risk the next 5 years, the government needs a coordinated and comprehensive plan that the ideal condition for disaster management.

An all-hazards approach is mainly considered in general disaster planning, with emergency management functions being divided into four areas: mitigation, preparedness, response, and recovery

Multidiscipline Approach
In hospital disaster management, the response to an incident and the actions to be taken in the pre and post-incident periods need to be defined

Most hospital disaster plans are primarily intended to treat and cure victims, and are developed by doctors and nurses. A multidisciplinary approach is essential in order to produce a plan that covers all aspects.

integrated approach should be developed by a task force composed of representatives from at least the engineering, security and prevention and nursing departments, in addition to the hospital disaster coordinator and medical director. A disaster coordinator with specific knowledge and skills will guide the organization towards an efficient and effective disaster response and an overall healthcare response.

Preparedness
Specific emergency procedures must then be developed to prevent or to contain the incident and thus minimize possible damage. Each specific risk/hazard may need a specific emergency procedure. Hospital should have a structure for standard operating procedure, as a guideline for creating each specific emergency procedure. A general procedure must be developed for alerting and activating staff reinforcement, specific logistics, and incident management hierarchy (structure). An alarm monitoring centre should be manage by personnel who are trained to receive calls from individuals observing potential crises or incidents.

The Hospital Incident Command System (HICS) is a organizational tool structured with main principles: early implementation, modular makeup and standardized terminology. Ideally, the first tier should include medical team, security personnel, and technical personnel, all of whom must be immediately available

should an incident arise. The first tier identifies the problem, initiates an appropriate emergency procedure. The second tier is called if the incident is, serious or if the first tier considers the incident to be beyond their capabilities. The second tier should consist of at least two staff-level personnel: one medical hospital incident manager, who focuses on the provision of appropriate incident medical care, and a hospital incident manager (non-medical), focuses on preserving hospital functionality and provides logistic and personnel support for the medical hospital incident manager.

Response

Four response plans must be available and ready for implementation: reception, evacuation, relocation and isolation plans.

A primary reception plan how to manage between patient inflow and existing resources. The aspects involved are triage, damage control therapy, and delayed therapy. A secondary reception plan focuses primarily on increasing the productivity of a specific hospital unit.

The evacuation plan also provides instructions for taking decisions on which patients to evacuate first. In operational terms, this is done by assessing important parameters

Recovery

The best overall result after an incident, it is important to evaluate communication and coordinate all aspects of incident and crisis management.

An integrated and multidisciplinary approach, includes preparedness risk assessment, response, and recovery, and these all involve several disciplines. The response phase, incident-specific emergency procedures must limit human and material damage, surge capacity is an emerging key element for which reverse triage may offer a solution. Each Hospital Incident Manager System is hospital-specific, and a set of plans for efficient incident management appropriate for one hospital. The Hospital Incident Manager System, it is important that all tiers, and specifically incident managers, are absolutely sure of their authority, responsibility, and the functional demands imposed upon them.

CARDIAC RESUSCITATION

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ABSTRACT

Introduction

Sudden cardiac arrest is defined as the cessation of effective cardiac mechanical activity as confirmed by the absence of signs of circulation. Sudden cardiac arrest is the most common fatal manifestation of cardiovascular disease and a leading cause of death worldwide. The exact incidence of sudden cardiac arrest is unclear, but in the United States alone, it has been estimated to be as high as 450,000 persons annually. Approximately 21% to 25% of sudden cardiac arrest events are due to pulseless ventricular arrhythmias (i.e., ventricular fibrillation/VF or pulseless ventricular tachycardia/VT), whereas the rest can be attributed to other cardiac rhythms (i.e., asystole or pulseless electrical activity/PEA).

Patients who suffer cardiac arrest due to VF or VT have a much higher chance of surviving the event compared with patients who present with PEA/asystole. The prognosis is better in patients with ventricular arrhythmias because (1) ventricular arrhythmias are potentially treatable with defibrillation (i.e., "shockable" initial rhythm) to restore circulation, whereas the other initial rhythms are not, and (2) ventricular arrhythmias are typically a manifestation of a cardiac etiology of cardiac arrest (e.g., acute myocardial infarction), whereas the other initial rhythms are more likely to be related to a noncardiac etiology and perhaps an underlying condition that is less treatable. Clinical outcomes for cardiac arrest are poor. Approximately 11% of out-of-hospital cardiac arrest (OHCA) and 20% of in-hospital cardiac arrest (IHCA) patients survive to hospital discharge.

The basic principles of resuscitation are an integral part of training for many health care providers (HCPs). Because timely interventions for cardiac arrest victims have the potential to be truly lifesaving, it is especially important for critical care practitioners to have a sound understanding of the evaluation and management of cardiac arrest. A number of critical actions *(chain of survival)* must occur in response to a cardiac arrest event.

Figure 1.1 The American Heart Association chain of survival paradigm.This figure represents the critical actions needed to optimize the chances of survival from cardiac arrest. The links (from left to right) include (1) immediate recognition of cardiac arrest and activation of the emergency response system; (2) early and effective cardiopulmonary resuscitation; (3) defibrillation (if applicable); (4) advanced cardiac life support; and (5) post–cardiac arrest care (including target temperature management if appropriate)

The chain of survival paradigm (Fig. 1.1) for the treatment of cardiac arrest remained unchanged in the 2015 American Heart Association (AHA) Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care from 2010 and is similar to other cardiac arrest guidelines. The chain of survival paradigm consists of five separate and distinct elements: immediate recognition that cardiac arrest has occurred and activation of the emergency response system; application of effective cardiopulmonary resuscitation (CPR); early defibrillation (if applicable); advanced cardiac life support; and initiation of postresuscitation care (e.g., targeted temperature management). When the chain of survival is implemented effectively survival for VF, OHCA can exceed 45%.

Cardiopulmonary resuscitation (CPR) was conceived as a temporary circulatory support procedure for otherwise healthy patients suffering sudden cardiac death. In most cases, coronary ischemia or primary arrhythmia is the inciting event. Since its inception, however, CPR use has been expanded to nearly all types of patients who suffer an arrest. A general approach currently recommended by the American Heart Associated is presented in Figure 1.2.

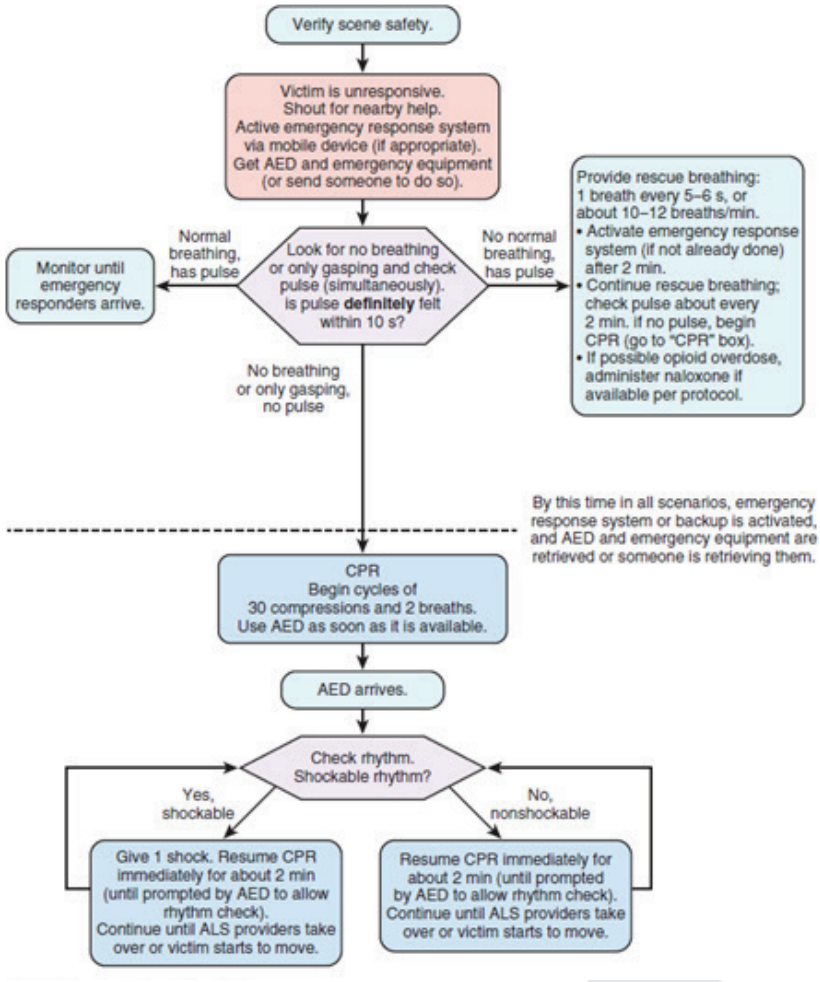

Figure 1.2 American Heart Association adult Basic Life Support (BLS) algorithm. AED, Automated external defibrillator; ALS, advanced life support; CPR, cardiopulmonary resuscitation. (From Kleinman ME, Brennan EE, Goldberger ZD, et al. Part 5: adult basic life support and cardiopulmonary resuscitation quality: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2015;132(18 suppl 2):S414–S435.)

Principles of Resuscitation

Current expert recommendations for resuscitation are much simpler than those in the past and stress the importance of effective circulatory support and prompt shock of pulseless VT and VF while de-emphasizing respiratory support.

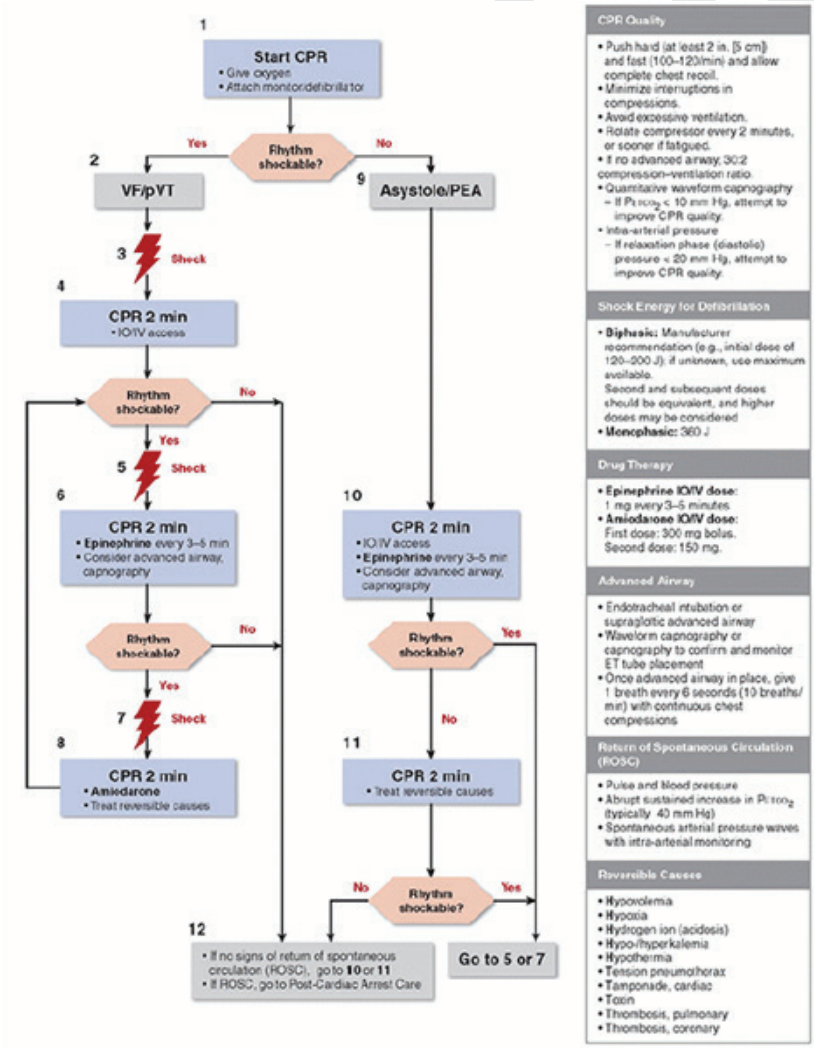

Figure 1.3. General overview of approach to cardiac arrest. CPR, cardiopulmonary resuscitation; IO, intraosseous; IV, intravenous; PEA, pulseless electrical activity; PETCO2, end tidal PCO2; PVT, pulseless ventricular tachycardia; ROSC, return of spontaneous circulation; VF, ventricular fibrillation.

Although that advice makes sense for most out of hospital events, in the hospital, the resuscitation team must quickly consider the specific circumstances of each arrest to determine the best course of action. For example, a mechanically ventilated patient found in VF will not be saved by a formulaic approach to arrhythmia treatment if it is not recognized that the cause of the event is a tension pneumothorax or major airway obstruction. Because survival declines exponentially with time after arrest (Fig. 1.4), most successfully resuscitated patients are revived in less than 10 minutes. To this end, first responders should summon help and begin effective chest compression. If the cardiac rhythm can be monitored and is pulseless VT or VF, unsynchronized direct current (DC) cardioversion using maximal energy should be delivered as quickly as possible. If these initial actions are unsuccessful, more prolonged, "advanced" resuscitation measures may be indicated

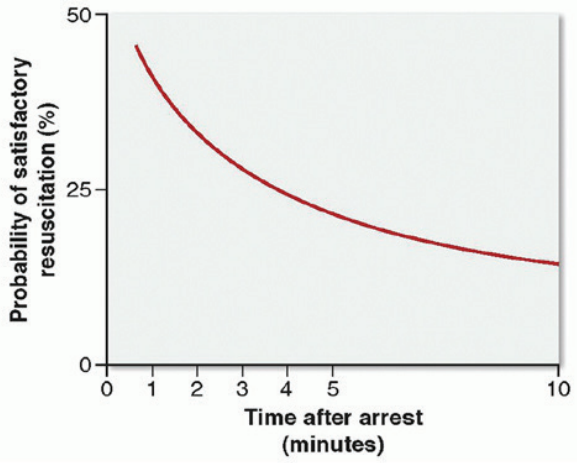

Figure 1.4. Probability of successful initial resuscitation after cardiopulmonary arrest. Exponential declines in survival result in low success rates after 6 to 10 minutes of full arrest conditions. The primary activities of resuscitation include (1) team direction, (2) circulatory support, (3) cardioversion/defibrillation, (4) airway management and ventilation, (5) establishing intravenous access, (6) administering drugs, (7) performance of specialized procedures (e.g., pacemaker and chest tube placement), and (8) database access and recording. Managing a cardiopulmonary arrest usually requires several persons to directly execute procedures. Additional personnel are needed for nonprocedural tasks such as documentation, chart review, and communication with the laboratory or other physicians, but limiting the number of people involved to the minimum required avoids confusion.

Principle 1: Define the Team Leader

A single person must direct the resuscitation team because chaos often surrounds the initial response. This person should attempt to determine the cause of the arrest, confirm the appropriateness of resuscitation, establish treatment priorities, and coordinate the steps of ACLS protocol. The leader should also monitor the electrocardiogram (ECG), order medications, and direct the actions of the team members but must avoid distraction from the command role by performing other functions.

Principle 2: Establish Effective Artificial Circulation

Blood flow during closed-chest CPR likely occurs by two complementary mechanisms: direct cardiac compression and thoracic pumping. First, compressions generate positive intracardiac pressures, simulating cardiac chamber contraction with the unidirectional heart valves helping to ensure forward flow. In addition, as the chest is compressed, a positive gradient is established between intrathoracic relative to extrathoracic arterial pressures, propelling flow forward. Retrograde venous flow is prevented by jugular venous valves and functional compression of the inferior vena cava at the diaphragmatic hiatus. On relaxation of chest compression, falling intrathoracic pressures promote blood return into the right heart chambers and pulmonary arteries, filling these structures for the next compression. Thus, when CPR is performed for more than 10 to 15 minutes, hypoperfusion predictably results in tissue acidosis. If performed improperly, CPR is not only ineffective but potentially injurious. Several points of technique deserve emphasis. Maximal flow occurs with a compression rate of 100 to 120 beats/min. Current recommendations have increased the ratio of compressions to breaths in an attempt to maximize flow. For the same reason, current protocols suggest continuing CPR for several minutes after electrical shock attempts. To optimize cardiac output, it is important to adequately compress the chest.

During CPR, it is difficult to determine whether blood flow is adequate, because pulse amplitude, an index of pressure, does not directly parallel flow and organs vary with regard to the flow they receive at a given pressure. For example, brain flow relates to differences between mean aortic pressure and right atrial pressure, assuming normal intracranial pressure. Therefore, increasing right atrial pressure will decrease brain blood flow when mean arterial pressure is held constant. Coronary blood flow, on the other hand, is best reflected by the diastolic aortic to right atrial pressure gradient. For both, vasoconstrictive drugs (i.e., epinephrine) are recommended to raise the mean aortic pressure.

Principle 3: Establish Effective Oxygenation and Ventilation

Establishing a secure airway and provision of supplemental oxygen are essential if the primary problem was respiratory in origin, or whenever resuscitative efforts continue for more than a few minutes. Except in unusual circumstances, ventilation can be quickly accomplished in the nonintubated patient with mouth-to-airway or bagmask ventilation. Because position, body habitus, and limitations of available equipment often compromise either upper airway patency or the seal between the mask and face, effective use of bag-mask ventilation often requires two people. When the airway is patent, the chest should rise smoothly with each inflation. Gastric distension and vomiting may occur if inflation pressures are excessive. Inflation pressures generated by bagmask ventilation are sufficient to cause barotrauma and impede venous return; to minimize these risks, breaths should be delivered slowly, avoiding excessive inflation pressures and allowing complete lung deflation between breaths. In cardiopulmonary arrest, the most common cause of airway compromise is obstruction of the upper airway by the tongue and other soft tissues. Thus, in most cases after effective chest compression and ventilation have been achieved, an experienced person should intubate the airway. As a rule, intubation attempts should not interrupt ventilation or chest compression for longer than 30 seconds. Therefore, all materials, including laryngoscope, endotracheal (ET) tube, and suction equipment, should be assembled and tested before any attempt at intubation. Inability to establish effective oral or bag-mask ventilation signals airway obstruction and should prompt an immediate intubation attempt. When neither intubation nor effective bag-mask ventilation can be accomplished because of abnormalities of the upper airway or restricted cervical motion, temporizing measures should be undertaken while preparations are made to create a surgical airway. The laryngeal mask airway (LMA) is an easily inserted, highly effective temporizing device. It is important to have an LMA, which is appropriately sized for the patient. If the LMA

is too large, it may obstruct the larynx or cause trauma to laryngeal structures. An LMA that is too small or inserted improperly may push the base of the tongue posteriorly and obstruct the airway.

In the arrest setting, direct visualization of the tube entering the trachea, symmetric chest expansion, and auscultation of airflow distributed equally across the chest (without epigastric sounds) are the most reliable clinical indicators of successful intubation. Colorimetric CO2 detectors attached to the ET tube may support impressions of proper tracheal tube placement; however, because circulation and CO2 delivery to the lungs are both severely compromised during CPR, detectors may fail to change color on many properly placed tubes. For the same reason, attempts to eliminate CO2 by ventilation are relatively ineffective. During CPR, ventilation should attempt to restore arterial pH to near-normal levels and provide adequate oxygenation. Unfortunately, the adequacy of ventilation and oxygenation is difficult to judge because blood gas data are rarely available in a timely fashion. Furthermore, blood gases alone are poor predictors of the outcome of CPR, making their use in decisions to terminate resuscitation of questionable value. The cornerstone of pH correction is adequate ventilation after effective circulation has been achieved.

Principle 4: Establish a Route for Medication Administration

Access to the circulation must be established rapidly during CPR. Existing peripheral IV catheters are perfectly acceptable for medication administration. When medications are given through peripheral IV lines, they should be followed by at least 20 mL of fluid to facilitate drug entry into the circulation and to prevent mixing incompatible drugs. Central venous catheters (CVCs) reliably deliver drugs directly to the heart, but valuable time should not be wasted inserting a CVC if functioning peripheral venous access exists. (There is also a theoretical concern of delivering very high drug concentrations close to the heart when using a CVC.) Femoral access is less desirable than a jugular or subclavian route because of the higher risk of infection, but is certainly easier to establish without interrupting CPR. A large intraosseous (IO) needle can be placed very rapidly into the marrow of a long bone, typically the proximal tibia. This IO access is an effective route for drug administration in those patients who do not have a functioning IV. The luxuriant venous plexus of bone provides an efficient conduit to the circulation. There are currently several commercially available stylet/needle devices to rapidly achieve IO access. Typically, the needle penetrates the cortex using a screwing motion until resistance fades. After removal of the stylet, IO positioning is confirmed by aspiration of a small amount of marrow and the ability to gravity-infuse fluid at a slow rate. Major advantages of the IO route include a high success rate for cannulation (>80%), quick insertion (<2 minutes), avoidance of CVC-related complications, and rapid delivery of drug to the circulation. (In experimental models, IO-administered drugs reach the heart in <30 seconds.) Risks are uncommon and predictable. These include nerve or vessel injury, extravasation of drug into soft tissue with necrosis, compartment syndrome, and osteomyelitis.

The intratracheal (IT) route may be used to produce therapeutic drug levels rapidly during resuscitation. Drugs given via the IT route must be delivered with at least 20 mL of fluid to permit most of the dose to access the alveolar compartment, where absorption occurs. The doses of all drugs given by the IT route should be increased at least 2 to 2.5 times than used with IV dosing. The IT route has been demonstrated to be effective for administration of naloxone, atropine, vasopressin, epinephrine, and lidocaine, easily remembered as by mnemonic "NAVEL." Some commonly used drugs (e.g., norepinephrine, CaCl2, NaHCO3) should not be given via the IT route. The first two agents may cause lung necrosis and the third inactivates surfactant.

Intracardiac injections, although dramatic, are rarely necessary, often unsuccessful, and offer no greater likelihood of successful resuscitation. In addition, intracardiac injections are fraught with complications

including coronary laceration, pneumothorax, and tamponade. Intramural drug injection may expose the myocardium to massive concentrations of vasoactive drugs, provoking intractable ventricular arrhythmias.

Principle 5: Create an Effective Cardiac Rhythm

As a conceptual guide to treatment, cardiac electrical activity during the arrest can be thought of in two broad categories. The first is the combination of pulseless ventricular tachycardia and ventricular fibrillation (VT/VF),and the second group consists of asystole and pulseless electrical activity (PEA).

Ventricular Tachycardias and Ventricular Fibrillation

VT and VF are the most commonly discovered rhythms in victims of sudden cardiac death. Although VF may be the original arrhythmia, in many cases, the first dysfunctional rhythm is VT, which deteriorates to VF as the heart becomes progressively hypoxic. VT is described as either pulseless or pulse generating. VT without a pulse is treated as VF. VT has been further subclassified as being either monomorphic or polymorphic because there are potential treatment implications for the polymorphic variety. Monomorphic VT is typically a monotonous appearing wide complex tachycardia with a constant axis. Torsades de pointes is the name given to a unique appearing form of polymorphic VT that is frequently associated with baseline prolongation of the QT interval. Torsades is characterized by a constantly changing QRS axis that produces an apparent "twisting of points" about the isoelectric axis. Many reversible precipitating factors have been identified, including hypomagnesemia and the use of tricyclic antidepressants, haloperidol, droperidol, type la antiarrhythmics (e.g., quinidine, procainamide, and disopyramide), and quinolone antibiotics. When VT/VF is encountered, the American Heart Association recommends consideration of a standard list of reversible causes including hypovolemia, hypoxia, acidosis, hypokalemia, hyperkalemia, and hypothermia (the "Hs") to go along with the ("Ts") tension pneumothorax, cardiac tamponade, toxins, pulmonary thrombosis, and coronary thrombosis. Both VT and VF potentially can be converted with electrical shock, but VF tends to be more resistant. Epinephrine is sometimes successful in coarsening a fine VF waveform prior to the attempted shock. Regardless of whether the initial rhythm is VF, or monomorphic or polymorphic VT, maximal intensity (360 J) unsynchronized *monophasic* shock should be administered as quickly as possible for all patients in VF and pulseless VT. (Equivalent lower-intensity biphasic [200 J] shocks are equally effective.)

For patients receiving open-chest defibrillation, epicardial shocks of 10 to 20 J are almost always sufficient. The goal of delivery for DC countershocks is to abolish all chaotic ventricular activity, allowing an intrinsic pacemaker to emerge. Many defibrillators allow a "quick look" at the rhythm before shock is attempted, but careful inspection of the rhythm is not mandatory before proceeding. Current guidelines recommend simple administration of single shocks. Defibrillators are typically calibrated to discharge through impedance less than that of the adult chest. Therefore, the delivered energy usually is lower than is indicated by the nominal machine settings. This is particularly true in situations, which increase the distance between the paddles and the heart, like morbid obesity and conditions producing high lung volumes (e.g., COPD, large tidal volumes, high PEEP). Improper paddle positioning also dissipates energy and reduces the rate of successful defibrillation. Using the anterolateral technique, paddles are placed at the cardiac apex and just below the clavicle to the right of the sternum. Because bone and cartilage are poor conductors of electricity, paddles should not be located over the sternum. Defibrillator paddles should not be placed over ECG monitor leads, implanted pacemakers or defibrillators, or transcutaneous drug patches, because of the possibility of electrical arcing and equipment damage. Contact between the defibrillator and chest wall should be maximized by use of conducting gels or pads. Standardsized (8 to 13 cm diameter) paddles on adult defibrillators provide optimal impedance matching between machine and chest wall.

The availability of AEDs has changed defibrillation from an often delayed procedure performed by an expert in a hospital or ambulance to one rapidly accomplished by a novice in a public location. Fortunately, considerable standardization of AEDs has occurred so that regardless of manufacturer, the same basic steps are always used: power on the defibrillator, attach the pads and connect the cables using the illustrations provided, wait for the device to analyze the rhythm and charge, make sure all people are clear of the patient, and then discharge the device if the machine advises to do so. Pulseless VT or VF that remains resistant to cardioversion after several minutes of effective CPR portends a poor outcome. If initial attempts at defibrillation prove unsuccessful, "coarsening" the rhythm and increasing the vascular tone with epinephrine (1 mg IV, every 3 to 5 minutes) may be helpful. All the while, effective ventilation and chest compression should be maintained. After epinephrine is given, maximum energy defibrillation should be repeated. When the preceding measures fail, a trial of the antiarrhythmic amiodarone (300 mg IV) may help convert the rhythm when followed by additional shocks.

The small subgroup of patients with *torsades* deserves special mention. Although torsades is not particularly resistant to cardioversion, the arrhythmia frequently recurs within a short time. For long-term control, discontinuation of potentially precipitating drugs and correction of electrolyte abnormalities are indicated. For patients with a previously normal QT interval, coronary ischemia is a common precipitant amenable to standard treatment. β-Blockers, lidocaine, and amiodarone have all been tried for refractory torsades without any one emerging as a clearly superior agent. For patients known to have prolonged baseline QT interval, MgSO4 may be helpful, but the most effective measure is to shorten the QT interval, usually by increasing the heart rate (i.e., pacing or catecholamine infusion).

Asystole and Pulseless Electrical Activity

For purposes of resuscitation, asystole and PEA are grouped together. Almost any rhythm is preferable to asystole, the complete absence of electrical activity (a flat ECG), but some rhythms (i.e., pulseless slow bradycardia or ventricular escape beats) are not much better. Therefore, a key aim in asystole is to stimulate some electrical activity and then modify that activity to a rhythm with a pulse. Because asystole usually indicates extended interruption of perfusion and carries a grave prognosis, its discovery should prompt serious consideration of whether resuscitative efforts should even begin. It makes no sense to countershock the truly asystolic patient because there is no "rhythm" to modify. Epinephrine (1 mg IV, every 3 to 5 min) given during effective CPR may restore a vestige of electrical activity. Manipulation of electrolyte balance (Ca2+, K+) also may be useful in specific cases. NaHCO3 may be useful if severe acidosis, hyperkalemia, or tricyclic antidepressant overdose is the cause of asystole. PEA, also known as electromechanical dissociation (EMD), is characterized by the inability to detect a pulse despite coordinated ECG complexes. Mechanical obstruction to the normal transit of blood through the heart may also cause PEA. Hence, atrial myxoma, mitral stenosis, and critical aortic stenosis may be potential causes.

Other reversible conditions that can produce this syndrome include (1) hypovolemia, particularly from acute blood loss (vasopressors lose effectiveness); (2) pericardial tamponade, suspected on the basis of venous engorgement, a history of chest trauma, or preexisting pericardial disease; (3) tension pneumothorax; (4) dynamic hyperinflation (auto-PEEP) from overly zealous ventilation; (5) massive pulmonary embolism by clot or air (thromboembolism may fragment and migrate during CPR, opening the central pulmonary artery and reestablishing effective output; air embolism can be treated by positioning the patient (left side down, Trendelenburg position) and/or transvenously aspirating air from the right heart); (6) hyperkalemia; and/or (7) metabolic acidosis. As adequate intravascular volume is assured or addressed, epinephrine is given in doses identical to those used for asystole.

Principle 6: Evacuate the Patient to the ICU as Soon as Practical

When cardiac arrests occur outside an ICU, facilities, equipment, and personnel for resuscitation are less than ideal. On general wards and in public hospital areas, it is often difficult to access the patient, especially if they have fallen alongside a bed or are in a bathroom or elevator. Simply getting emergency equipment to the patient's side can be a challenge in cramped quarters. There is often a crush of unhelpful bystanders and distraught family members, and even the patient's primary caregiver's effectiveness is hindered by their shock from an unexpected arrest. Electrical access and suction capabilities are commonly limited and specialized equipment, especially for airway management, is not always available. However, the most important limitation of performing resuscitation outside the ICU, especially in a remote part of the hospital (e.g., CT scanner), is that many of the personnel available to help have little experience performing real resuscitations. Preparation of emergency medications and assistance with procedures that are second nature for ICU personnel are often unfamiliar to non-ICU workers. For all these reasons it makes sense to do the absolute minimum required to establish ventilation and a rhythm that produces a pulse, then transport the patient to the ICU.

Principle 7: Reevaluate and Stabilize

After arriving in the ICU with a perfusing rhythm with adequate oxygenation and ventilation, it is important to rethink the cause of the arrest, to take measures to prevent recurrence, and to search for resuscitation complications. Tubes and catheters inserted during resuscitative efforts are often suboptimally positioned or are inserted with less than ideal sterile technique. Any intravenous catheter not known to be inserted in a sterile manner should be removed altogether or, if still needed, replaced at a new site using sterile technique. The position of the ET tube and any chest tubes or CVCs should be confirmed radiographically. (It is extremely common that emergently inserted ET tubes have been advanced into the right main bronchus.) The chest radiograph should also be examined for evidence of resuscitation or procedural injury (e.g., hemothorax or pneumothorax or rib or sternal fractures) and for clues to the cause of the original arrest (mediastinal widening of aortic injury, enlarged cardiac silhouette of pericardial tamponade, pneumothorax). The chest film should also be evaluated for the presence of aspiration or pneumonia that may have precipitated the arrest or resulted from it. If there is a suspicion of hemothorax, or hemoperitoneum, or retroperitoneal hematoma, chest and abdominal CT scans are usually diagnostic. However, careful consideration should be given to transporting a recently resuscitated patient outside the ICU; potential benefits should clearly outweigh the risks. If there is suspicion that the arrest may have been precipitated by a neurological event (e.g., ischemic stroke, hemorrhage, tumor, new seizure), it is prudent to obtain a noncontrast head CT scan with the same caveats regarding transport safety. For patients who are not fully awake after resuscitation, the prospect of ongoing seizures should be considered. If a seizure is a reasonable possibility, an electroencephalogram (EEG) should be obtained.

Acid-base and electrolyte abnormalities are so common after resuscitation that it makes sense to evaluate a full panel of electrolytes, especially Na+, K+, Ca2+, Mg3+, and an arterial blood gas. Because hypoglycemia can cause cardiac arrest, and post arrest hypoglycemia and hyperglycemia can cause or exacerbate brain injury, a rapid determination of blood glucose should be done. If there is suspicion that the cause of the arrest could be medication or toxin ingestion, obtaining a urine or plasma drug screen and specific drug levels (e.g., digitalis, lidocaine, phenytoin) may be enlightening. Although troponin and creatine phosphokinase (CPK) levels are frequently modestly elevated, they rarely provide a definitive diagnosis. Noteworthy elevation of the myocardial band (MB) isoenzyme of CPK is unusual unless repeated high-energy electrical shocks have been delivered.

Although elevations of white blood cell counts are routine, they are nonspecific and by themselves should not drive antibiotic use. A decision to obtain lung, blood, or urine cultures should be made on an individual basis, depending on the level of suspicion the role of infection played in the arrest. It is prudent to obtain a 12-lead ECG in all patients after stabilization to evaluate the rhythm and to look for signs of infarction, ischemia, and electrolyte abnormalities, conduction defects, and preexcitation pathways.
Finger oximetry should be utilized to maintain oxygen saturation at 94% to 96%. In general, patients should not be hyperventilated and hyperoxia must be avoided. Isotonic fluid should be given judiciously to treat hypotension, defined as systolic blood pressure less than 90 mm Hg. Typical fluids used in this setting are normal saline or lactated Ringers. Vasoactive drugs for the patient requiring catecholamine support are norepinephrine, epinephrine, and dopamine. The patient who is alert and able to follow commands should be monitored closely and further evaluated as described. Where patients cannot follow commands, careful temperature regulation as described below should be emphasized.

Principle 8: Preserve the Brain

Because neurological outcomes in survivors of cardiopulmonary arrest are poor, there has long been interest in methods for cerebral preservation. It should go without saying that maintaining a reasonable perfusion pressure and hemoglobin concentration and saturation are prerequisites for optimal cognitive recovery. The association of worse outcomes associated with hypoglycemia and hyperglycemia suggests that maintaining a normal range of glucose is helpful. There is no evidence to support the routine administration of anticonvulsants, anticoagulants, barbiturates, benzodiazepines, or neuromuscular blockers. Although unproven for this purpose, prevention of excessive cerebral metabolic demand (e.g., suppression of fever, seizures) makes sense and, particularly with respect to temperature control, is safe and inexpensive. The patient who remains comatose after cardiac arrest should have a temperature maintained in the range of 34°C to 36°C.

Perhaps, even more important, is the avoidance of fever. Potential candidates for this targeted temperature management approach should not have active bleeding or significant bradycardia because hypothermia may exacerbate both, as well as cause other complications. Therapeutic hypothermia is difficult if not impossible to achieve in waking patients without deep sedation and usually therapeutic neuromuscular blockade to prevent the inevitable, heat-generating shivering. Interestingly, external skin warming (e.g., by air blanket) may effectively block the shiver response as body temperature falls. Servoregulated intravenous cooling catheters have emerged as the current standard of practice. Regardless of method, the target is a core temperature of 36°C for 12 to 24 hours, with subsequent slow rewarming over 6 to 8 hours.

Summary

1. The success (hospital discharge without neurological impairment) of cardiopulmonary resuscitation is highly variable among patient populations. Cardiopulmonary resuscitation is very effective when applied promptly to patients with sudden cardiac death because of electrical instability, but is quite ineffective when applied in chronically debilitated patients and those suffering arrest as part of the natural progression of multiple organ failure.
2. The goal of resuscitation is to preserve neurological function by rapidly restoring oxygenation, ventilation, and circulation to patients with arrested circulation.
3. The resuscitation status of every patient admitted to the ICU should be considered at admission. When a clear determination regarding resuscitation status cannot be made quickly, the physician generally should on the side of promptly initiating resuscitation efforts.

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PREPARING FOR NATURAL DISASTER IN AIRWAY DISEASES: ASTHMA

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ABSTRACT

Natural disasters may impact on pre-existing chronic respiratory problems, for example via loss of power for those requiring oxygen or nebulizations, by destroying health services and infrastructural and and by removing access hospitals and clinics, for example, for physiotherapy and early intervention for disease exacerbation.

Disaster preparedness and respiratory disease

Respiratory diseases are common after natural disasters. The types of medications and equipment required to be stockpiled for a natural disaster is less clear, but are often focused on Acute Respiratory Infection (ARI). Various reviews have highlighted that the medications required should be a mix of pharmaceutical agents designed to treat both the acute respiratory infections, which are common after many types of disasters, and exacerbations of chronic respiratory diseases. Following Hurricane Andrew in 1992, field hospitals were reporting depletion of all their antibiotics within 24 h. Drugs for more chronic respiratory diseases, including asthma and chronic obstructive pulmonary disease, become increasingly important, particularly as the ARI recede. Following the eruption of Mt Pinatubo in the Philippines, the majority of medications dispensed to the 20000 evacuees were for chronic medical conditions.

Bronchodilators may be required acutely for exacerbations of asthma or isolated cases of bronchospasm, which may be related to smoke inhalation from bushfires or other sources. A review of data from the 2004 National Hospital Ambulatory Medical Care Survey in the USA identified that the most likely chronic respiratory disease requirements for a disaster were bronchodilators, oral steroids and antibiotics.

The stockpiling of ventilators, particularly for traumatic injury post disaster, is certainly recommended, although little is known about the success of such stockpiling. There will be a number of roles for these ventilators, including movement from the scene, movement between hospitals and in-hospital care, so there should have a mix of both transport and critical care ventilators.

What should you put in a disaster kit?

The American Red Cross recommends including a seven-day supply of medicine in your disaster kit. Keep track of the expiration dates of the medicines inside the kit, starting with the medicine that expires first. This can be done by listing the contents of the kit on an index card with the help of the pharmacist.

ASTHMA

Asthma exacerbation is a major cause of morbidity, can need acute care and result in death during natural disasters. Treatment for exacerbation needs a rapid evaluation of patients. Most asthmatics will have medications that control asthma but their need for acute care develops in the presence of smog, dust, ashfall and poor air quality after disasters. Even stress in itself can trigger an exacerbation.

A study used an Asthma Control Test **(ACT)**, a brief and patient-based tool to evaluate asthma control. Significant decreases were observed for the items:

"Asthma keeps you from getting much done at work"
"Shortness of breath"
"Asthma symptoms wake you up
"Patient rating of control"

ACT, an easy and practicable tool clearly demonstrated the asthma exacerbation in evacuation shelters without a lung function testing. **ACT** may contribute to the management of health crisis not only for East Japan but for the other forthcoming unavoidable disasters.

Stress & Asthma

in the United States, the economically disadvantaged and some ethnic minorities are often exposed to chronic psychosocial stressors and disproportionately affected by asthma. Current evidence suggests a causal association between chronic psychosocial stress and asthma or asthma morbidity. Recent findings suggest potential mechanisms underlying this association, including changes in the methylation and expression of genes that regulate behavioral, autonomic, neuroendocrine, and immunologic responses to stress. There is also evidence suggesting the existence of susceptibility genes that predispose chronically stressed youth to both post-traumatic stress disorder and asthma. Decades of research show that stressors, when perceived as threatening and unmanageable, modify the activity of the hypothalamic-pituitary-adrenocortical (HPA) axis and the autonomic nervous system (ANS). HPA activation occurs when neurons in the paraventricular nucleus of the hypothalamus secrete corticotropin releasing hormone (CRH). This molecule travels through the hypophyseal portal circulation to the anterior pituitary gland, which responds to its presence by secreting a pulse of adrenocorticotropin hormone (ACTH). The ACTH signal is carried through the peripheral circulation to the adrenal glands, which synthesize and release cortisol in the zona fasciculata. The ANS consists of sympathetic and parasympathetic branches whose effector molecules include epinephrine, and norepinephrine, and acetylcholine. By changing the outflow of these systems, stress alters the systemic balance of glucocorticoids and catecholamines, as well as concentrations of these (and other) hormones in primary and secondary lymphoid organs . Macrophages and lymphocytes have functional receptors for these hormones (glucocorticoid receptors for cortisol, alpha and beta adrenergic receptors for catecholamines), and ligation of those receptors alters these cells' repertoires of gene expression, with downstream implications for trafficking, signaling, proliferation and differentiation, and effector functions. Via these modulatory influences, chronic stressors potentiate reactivity to asthma triggers, e.g., allergens, infections, and in doing so may exacerbate airway inflammation and airflow obstruction.

Stress and Response to Treatment

Stress may increase asthma morbidity by reducing response to inhaled corticosteroids and inhaled beta2 agonists. Acute stress and chronic stress have been associated with reduced expression of the genes encoding the glucocorticoid receptor (by 5.5 fold) and the beta-2 adrenergic receptor (by 9.5 fold) in leukocytes of children with asthma (adjusted $P < 0.05$ in both instances). Several studies support the hypothesis that chronic stress leads to down-regulation of glucocorticoid receptor expression and function.

Climate change and respiratory diseases

Climate change represents a massive direct threat to respiratory health by promoting or aggravating respiratory diseases or indirectly by increasing exposure to risk factors for respiratory diseases. Climate

change represents a massive threat to respiratory health: 1) by directly promoting or aggravating respiratory diseases; or 2) by increasing exposure to risk factors for respiratory diseases. Climate change increases the amount of pollen and allergen produced by each plant, mould proliferation and the concentrations of outdoor ozone and particulate matter at ground level. The main diseases of concern are asthma, rhinosinusitis, chronic obstructive pulmonary disease (COPD) and respiratory tract infections. Groups at higher risk of climate change effects include individuals with pre-existing cardiopulmonary diseases or disadvantaged individuals.

Changes in meteorological parameters substantially increase respiratory morbidity and mortality in adult patients with common chronic lung diseases, such as asthma and COPD, and other serious lung diseases . Respiratory diseases similarly increase among children during heat waves . Extreme heat and high humidity trigger asthma symptoms . Cyclones have also been related to asthma. Cold weather, by increasing cold exposure, will increase overall respiratory infections in individuals with underlying COPD.

Thunderstorms occurring during the pollen season have been observed to induce severe asthma attacks in pollinosis patients . Associations between thunderstorms and asthma morbidity have been identified in multiple locations around the world . The thunderstorm-related epidemics are limited to late spring and summer when there are high levels of airborne pollen grains. There is a close temporal association between the arrival of a thunderstorm, a major rise in concentration of pollen grains and the onset of asthma epidemics. The most prominent hypotheses for thunderstorm-related asthma are linked to bio-aerosols and involve the role of rainwater in promoting the release of respirable particulate matter. After hydration and rupture by osmotic shock during the beginning of a thunderstorm, pollen grains release part of their cytoplasmic content into the atmosphere, including inhalable, allergen-carrying paucimicronic particles such as starch granules and other cytoplasmic components. As a consequence of climate change on pollen and on its effects in allergic patients, After hydration and rupture by osmotic shock during the beginning of a thunderstorm, pollen grains release part of their cytoplasmic content into the atmosphere, including inhalable, allergen-carrying paucimicronic particles such as starch granules and other cytoplasmic components.

Climate change will increase the frequency and intensity of floods and cyclones and thus fungal spore production, a powerful asthma and rhinitis trigger. The link between moulds and asthma and rhinitis is well-known and has been established through exposure to dampness and moisture in indoor environments as proxy of microbial agents. In addition, exposure to natural disasters, such as floods and cyclones, was reported to exacerbate the burden of depression, anxiety and stress that are risk factors for asthma . High stress for adults after weather-related disasters can have serious implications for children, by impeding the adults' ability as caregivers and, in extreme circumstances, resulting in neglect .

Respiratory doctors have a vital role in addressing climate change, just as they did with tobacco, by communicating how climate change is a serious, but remediable, hazard to their patients.
A presentation on basic pathogenesis of asthma and latest guidelines by GINA 2019 is included in the presentation.

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VICTIM OF DISASTER – IN PSYCHOLOGICAL PERSPECTIVE

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ABSTRACT

Introduction
Indonesia is an area prone to natural disasters. Volcanoes, earthquakes, tsunamis, and even combinations of several natural disasters can occur at any time. Being in the ring of fire area makes this country need to be vigilant about natural disasters that can happen at any time (www.id.undp.org). What about other countries? For countries with certain geographical conditions, other forms of natural disasters can also occur. Japan with its earthquake, Taiwan with its typhoon, even with America, precisely in California, with the occurrence of forest fires occurring quite routinely (www.worldatlas.com). Another disaster is the event of 911 attack in USA. Ever since, disaster preparedness and response has developed into a discrete subspeciality in medicine an national health priority (Neria, Galea, & Norris, 2009).

The occurrence of disasters will have an impact — both physical and non-physical. Effects could vary from a small scale up to a large scale (Burkle, 1999). However, the most challenging result is undoubtedly an impact on humans. Here, humans are victims. Depending on the level of the disaster, the impact on victims can also widely vary (Raphael, Singh, Bradbury, & Lambert, 1983).

Disaster
What is a disaster? Disaster is when the resource that is owned by an individual is far insufficient than the situation of change he is facing (www.ifrc.org; The American College of Emergency Physicians, in Zibulewsky, 2001). A person can be very disturbed by the floods for the first time of occurrence, but can also feel quite calm because they have already experienced flood quite often. However, if we discuss a disaster that has a large scale and even causes death, it seems that the resources that humans have are never enough.

In general, disasters are divided into two major types, namely natural disasters and human-caused disasters (Goldmann & Galea, 2013)-----. For the second type, it can be subdivided to intentional, and unintentional. Natural disasters are defined as major adverse events resulting from natural process of the earth (Cutter, Emrich, Mitchell, Boruff, Gall, Schmidtlein, & Melton (2015). Take, for example, the shift of the earth's plate that caused an earthquake, and then a tsunami like what happened in Indonesia in 2004. This was one example includes natural disasters. For some people who have heard of cases of nuclear reactor leaks at Chernobyl, 1986, or in Fukushima recently, or train collisions, including in unintentional man-made disasters. The last example is Genocide or mass shooting cases such as those that occurred in New Zealand in the first half of this year. These examples are included in deliberate man-made disasters.
Disasters, especially natural disasters, have become a concern for many countries that face various disaster situations. Each country has its task force that functions to mitigate disasters immediately after the disaster. Findings shows an increasing numbers of human impact from around 162 million people in 2005, to more than 330 million in 2010 (Goldmann & Galea, 2013). It is a high number that has to be taken care seriously.

Human as Victim of Disaster
When we try to understand humans as victims of disaster, many things can be discussed. Primarily, we can study the process of the meaning of disaster in the human mental system. What exactly it is, and why does

a disaster-affected individual experience psychological disorders, both mild and severe? As mentioned earlier, every human being has their resources to deal with life. Every human being, along with their personal development and life journey, has the capacity to cope with various stressful external situations. Disaster is the meaning of the occurrence of a major event that occurred the victim. Disasters in both natural and man-made disasters are something that cannot be predicted very precisely. Although steps to reduce the impact of disasters have been carried out, the magnitude of the impact of disasters cannot always be calculated correctly. This unpredictable situation certainly triggers stress in humans, in the end. And if we examine the emergence of stress, one of them begins because there is a discrepancy between what is expected and the facts that must be faced. Research by Siddiqi, Siddiqi, Saeed, & House (2005) found that impacts on victims not always decrease by time, therefore an intervention has to be started as soon as possible. In cases such as not being able to arrive on time to work due to congestion, for example, the stress experienced by someone is certainly different from those who experience the destruction of house and other property due to occurrence of landslides. The magnitude of this discrepancy gap determines how much the psychological impact on individuals in disaster situations.

Psychopathology after Disaster

What usually happens during and shortly after the disaster? During disasters, people generally experience panic. Not knowing what was happening, not being able to find a way to get out of the situation, or feeling about dying from a disaster that approached. If this individual finally survives this situation, when the disaster has passed, and the situation is more stable, then the following thoughts emerge. For example, the idea of how much loss occurs and whether it can be overcome alone. Then, thoughts about how his future will be with the impact of the disaster. Then if the impact occurs to cause good death from the surrounding environment, especially from the closest circle like the family, then plural to come up with thoughts about how to live without a family who died after this (Gordon-Hollingsworth, Yao, Chen, Qian, & Chen, 2015). All of these are big situations that are unpredictable and may be very different from what the individual thinks before a disaster strikes.

Albeit researches about post disaster psychopathology shows that most victims could bounce back to their normal life in a relatively short time after disaster, but there are also numbers of victims who cannot cope well with the post disaster situation. Experiencing a disaster triggers the emergence of certain psychological symptoms. We already know that panic is main response. Panic arises when the victim cannot know how to get out of an unpredictable situation. Being in a state of stress, of course, makes an individual in a state of stress. Then stress symptoms are the next ones to appear. According to Goldmann & Galea (2013), after the critical phase has passed, it is ubiquitous for victims to experience a situation of Post Traumatic Stress Disorder (PTSD). PTSD is characterized by re-rexperiencing the event through nightmares and/or flashbacks, avoiding stimuli that triggers memory about the events, also numbing in emotional responses and symptoms of hyperarousal. Second psychopathology that common shows after disaster is Major Depressive Disorder. A disorder that characterize by sadness and loss of pleasure or interest in things once enjoyed, as well as combination of changes in sleep an eat pattern, difficulty in concentration also highly irritated. Some other psychological symptoms as Generalized Anxiety Disorder (GAD), death anxiety, panic disorder and also phobias were reported shown as a result of disaster. Polemikou (2019) also stressed psychological trauma, as “normal” response to the “abnormal” situation. Psychological trauma not always occurs to victims experiencing the disaster, but also victims who might be in a safe place but witnessing it through media, like findings on research conducted to measure acute stress on Dutch after the MH17 crash (Jeronimus, Snippe, Emerencia, de Jonge, & Bos, 2018). Similar research on exposure of disaster via media also shows increasing level of PTSD (Hall, Xiong, Yip, Lao, Shi, Sou, Chang, Wang, & Lam, 2019).

Psychological Intervention

The handling of disaster victims at the earliest stage is PFA (Psychological First Aid), which is a psychological treatment that prioritizes the fulfillment of the victim's basic psychological condition. PFA is seen as the preferred strategy in handling the crisis phase of disaster victims (Goldmann & Galea, 2013). What are the PFAs? (1) Humane, supportive & practical assistance to fellow human beings who recently suffered a serious stressor; (2) Non-intrusive, practical care and support; (3) Assessing needs and concerns; (4) Helping people to address basic needs (food, water); (5) Listening, but not pressuring people to talk; (6) Comforting people and helping them to feel calm; (7) Helping people connect to information, services and social supports; (8) Protecting people from further harm, and; (9) Validate emotions.

Both psychological and medical interventions are important for disaster victims. Victims need to get an initial assessment of their intervention needs as soon as the disaster occurs. If medical conditions are more critical than psychological conditions, medical treatment must be prioritized. However, if medical conditions are not severe, psychological intervention with the aim of presenting a sense of security for the victim can be carried out. WHO already state an integrated mental health service to primary care services. With this system, victims could quickly being taken care of after disaster (Siddiqi, Siddiqi, Saeed, & House, 2005). Mental health workers and physicians can work together as a helping professional. As a helping profession that works in the context of a crisis, the main purpose of the intervention must be understood together. PFA is a formula that covers the techniques carried out in ensuring the condition of the victim. It is often forgotten that despite acting more in the medical realm, professionals who are also of medical background are also expected to be able to apply the PFA. Because in medical treatment, interactions and communication with victims also present, which may not be done by non-medical personnel due to limitation of medical skills.

PFA, as an underlying postulate, is based on fundamental communication techniques. Empathy is the core, which is supported by how we give active listening responses, paraphrase sentences and the victim's mind, to do emotional validation, all of which aim to keep the victim's condition psychologically from getting worse.

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ABSTRACT

Indonesia memiliki kondisi geografis, geologis, hidrologis, dan demografis yang memungkinkan terjadinya bencana, baik yang disebabkan oleh faktor alam, faktor non alam maupun faktor manusia yang menyebabkan timbulnya korban jiwa manusia, kerusakan lingkungan, kerugian harta benda, dan dampak psikologis.

Wilayah Indonesia secara geografis dan geologis,

- merupakan negara kepulauan yang terletak pada pertemuan empat lempeng tektonik, yaitu: lempeng Euroasia, Australia, Pasifik, dan Filipina.
- terdapat 130 gunung api aktif di Indonesia
- terdapat lebih dari 5.000 sungai besar dan kecil yang 30% di antaranya melewati kawasan padat penduduk dan berpotensi terjadinya banjir, banjir bandang dan tanah longsor pada saat musim penghujan.

Bencana dapat terjadi kapan saja dan dimana saja.
Beberapa kejadian bencana besar di Indonesia antara lain:

a. Gempa bumi dan tsunami.

- Gempa bumi dan tsunami terbesar tanggal 26 Desember 2004, melanda Provinsi Nanggroe Aceh Darussalam dan sebagian wilayah Provinsi Sumatera Utara dengan jumlah korban yang sangat besar, yaitu 120.000 orang meninggal, 93.088 orang hilang dan 4.632 orang luka luka.
- Tanggal 17 Juli 2006, peristiwa yang sama kembali melanda pantai Selatan Jawa (Pangandaran, Ciamis, Tasikmalaya, Garut, Banjar, Cilacap, Kebumen, Gunung Kidul dan Tulung Agung)
- Pada 25 Oktober 2010, peristiwa gempa bumi dan tsunami kembali terjadi di Kab Mentawai Provinsi Sumatera Barat

b. Gempa bumi.

Gempa bumi Nias, Sumatera Utara, DI Yogyakarta dan Jawa Tengah serta Sumatera Barat. Terbaru : Gempa bumi berkekuatan 7,7 SR mengguncang Kabupaten Donggala, Kota Palu, Sulawesi Tengah pada hari Jumat (28/9/2018),

c. Ledakan bom.

- Ledakan bom Bali I 12 Oktober 2002,
- ledakan bom Bali II 1 Oktober 2005 dan
- ledakan bom di wilayah Jakarta (bom Gereja Santa Anna dan HKBP, bom Plaza Atrium, bom sekolah Australia, bom tahun baru Bulungan, bom kompleks Mabes Polri 3, bom bandara Soekarno Hatta, bom JW Marriott, bom Pamulang Tangerang, bom di Hotel JW Marriott dan Ritz Carlton Jakarta)

d. Letusan gunung berapi.

Letusan Gunung Merapi di Jawa Tengah

e. Kegagalan teknologi.

Ledakan pabrik pupuk Petro Widada Gresik

f. Banjir lumpur panas.

Tahun 2006 : lumpur lapindo di Sidoarjo pengungsian sebanyak 10.574 jiwa;

g. Banjir bandang.

Banjir bandang di Kabupaten Teluk Wondama Provinsi Papua Barat

h. Konflik.

Sejak awal tahun 1999 telah terjadi konflik vertikal dan konflik horizontal di Indonesia, ditandai dengan timbullnya kerusuhan sosial, misalnya di Sampit Sambas, Kalimantan Barat, Maluku, Aceh, Poso, Sulawesi, Nusa Tenggara Timur, Papua, Tarakan dan berbagai daerah lainnya yang berdampak pada terjadinya pengungsian penduduk secara besar besaran.

Semua kejadian tersebut menimbulkan krisis kesehatan, antara lain: lumpuhnya pelayanan kesehatan, korban mati, korban luka, pengungsi, masalah gizi, masalah ketersediaan air bersih, masalah sanitasi lingkungan, penyakit menular, gangguan kejiwaan dan gangguan pelayanan kesehatan reproduksi.

Bencana tak hanya mengakibatkan warga kehilangan tempat tinggal dan anggota keluarganya saja, mesti waspada terhadap penyakit yang mengintai para pengungsi.
Beberapa korban selamat justru terkena pneumonia yang mereka sebut sebagai “tsunami lung”. nfeksi paru-paru itu muncul ketika para korban yang tersapu oleh gelombang tsunami menghirup air laut yang terkontaminasi dengan lumpur dan bakteri.

Berbagai upaya penanggulangan bencana yang dapat dilakukan pada setiap tahap dalam siklus bencana antara lain:

a. Pencegahan dan Mitigasi;
   Upaya ini bertujuan menghindari terjadinya bencana dan mengurangi risiko dampak bencana, antara lain:
   1) penyusunan kebijakan, peraturan perundangan, pedoman dan standar;
   2) pembuatan peta rawan bencana dan pemetaan masalah kesehatan
3) pembuatan brosur/leaflet/poster

4) analisis risiko bencana
5) pembentukan tim penanggulangan bencana
6) pelatihan dasar kebencanaan
7) membangun sistem penanggulangan krisis kesehatan berbasis masyarakat.

b. Kesiapsiagaan;
   Upaya kesiapsiagaan dilaksanakan untuk mengantisipasi kemungkinan terjadinya bencana. Upaya kesiapsiagaan dilakukan pada saat bencana mulai teridentifikasi akan terjadi. Upaya upaya yang dapat dilakukan antara lain:
   1) penyusunan rencana kontinjensi;
   2) simulasi/gladi/pelatihan siaga;
   3) penyiapan dukungan sumber daya;
   4) penyiapan sistem informasi dan komunikasi.

c. Tanggap darurat;
   Upaya tanggap darurat bidang kesehatan dilakukan untuk menyelamatkan nyawa dan mencegah kecacatan, antara lain:
   1) Penilaian cepat kesehatan (rapid health assessment);
   2) Pertolongan pertama korban bencana dan evakuasi ke sarana kesehatan;
   3) Pemenuhan kebutuhan dasar kesehatan;
   4) Perlindungan terhadap kelompok risiko tinggi kesehatan.

d. Pemulihan.
   Upaya pemulihan meliputi rehabilitasi dan rekonstruksi.
   Upaya rehabilitasi bertujuan mengembalikan kondisi daerah yang terkena bencana yang serba tidak menentu ke kondisi normal yang lebih baik. Upaya rekonstruksi bertujuan membangun kembali sarana dan prasarana yang rusak akibat bencana secara lebih baik dan sempurna.
   Upaya-upaya yang dilakukan antara lain:
   1) perbaikan lingkungan dan sanitasi;
   2) perbaikan fasilitas pelayanan kesehatan;
   3) pemulihan psiko sosial;
   4) peningkatan fungsi pelayanan kesehatan;

Gambar 2.1. Siklus Penanggulangan Bencana (hal 7 buku Pedoman Tehnis Penanggulangan Krisis Kesehatan akibat bencana)

Rencana kontinjensi:
Suatu proses perencanaan ke depan, dalam keadaan yang tidak menentu, di mana skenario dan tujuan disepakati, tindakan teknis dan manajerial ditetapkan, dan sistem tanggapan dan pengerahan potensi disetujui bersama untuk mencegah, atau menanggulangi secara lebih baik dalam situasi darurat atau kritis.

Penilaian cepat masalah kesehatan (Rapid Health Assessment, RHA) :
Serangkaian kegiatan yang meliputi pengumpulan informasi subjektif dan objektif guna mengukur kerusakan dan meng-identifikasi kebutuhan dasar penduduk yang menjadi korban dan memerlukan ketanggapdarurat-an segera. Kegiatan ini dilakukan secara cepat karena harus dilaksanakan dalam waktu yang terbatas selama atau segera setelah suatu kedaruratan. Tim yang dapat diberangkatkan bersamaan

dengan Tim Reaksi Cepat

Tim Reaksi Cepat (TRC):
Tim yang sesegera mungkin bergerak ke lokasi bencana setelah ada informasi bencana untuk memberikan pelayanan kesehatan bagi korban.

Tim Bantuan Kesehatan:
Tim yang diberangkatkan untuk menangani masalah kesehatan berdasarkan laporan Tim RHA.

Manajemen penanggulangan bencana adalah pengelolaan penggunaan sumber daya yang ada untuk menghadapi ancaman bencana dengan melakukan perencanaan, penyiapan, pelaksanaan, pemantauan dan evaluasi di setiap tahap penanggulangan bencana yaitu pra, saat dan pasca bencana.

Perlu mengenal karakteristik setiap ancaman, sehingga dapat menyusun langkah langkah pencegahan, mitigasi dan kesiapsiagaan termasuk dalam penyusunan rencana operasional saat terjadi bencana.

Manajemen penanggulangan bencana, menjadi penting karena :
- Nyawa dan kesehatan masyarakat merupakan masalah utama;
- Waktu untuk bereaksi yang sangat singkat;
- Risiko dan konsekuensi kesalahan atau penundaan keputusan dapat berakibat fatal;
- Situasi dan kondisi yang tidak pasti;
- Petugas mengalami stres yang tinggi;
- Informasi yang selalu berubah.

Pada saat terjadi bencana perlu adanya mobilisasi SDM kesehatan yang tergabung dalam suatu Tim Penanggulangan Krisis yang meliputi:
- Tim Reaksi Cepat/TRC;
- Tim Penilaian Cepat/TPC (RHA team);
- Tim Bantuan Kesehatan.

Sebagai koordinator tim adalah Kepala Dinas Kesehatan Provinsi/Kabupaten/Kota (sesuai Surat Kepmenkes Nomor 066 tahun 2006).

1) Tim Reaksi Cepat
   Tim yang diharapkan dapat segera bergerak dalam waktu 0–24 jam setelah ada informasi kejadian bencana. Kompetensi TRC disesuaikan dengan jenis bencana spesifik di daerah dan dampak kesehatan yang mungkin timbul. Sebagai contoh untuk bencana gempa bumi dengan karakteristik korban luka dan fraktur, kompetensi TRC terdiri dari :
   a) pelayanan medik;
      - dokter umum
      - dokter spesialis bedah/orthopedi
      - dokter spesialis anestesi
      - perawat mahir (perawat bedah, gadar)
      - tenaga Disaster Victims Identification (DVI)
      - apoteker/tenaga teknis kefarmasian
      - sopir ambulans
   b) surveilans epidemiolog/sanitarian;

c) petugas komunikasi;
d) petugas logistik.

2) Tim Penilaian Cepat (RHA team)
Tim yang bisa diberangkatkan dalam waktu 0 24 jam atau bersamaan dengan TRC dan bertugas melakukan penilaian dampak bencana dan mengidentifikasi kebutuhan bidang kesehatan, minimal terdiri dari:
a) dokter umum
b) epidemiolog
c) sanitarian

3) Tim Bantuan Kesehatan
Tim yang diberangkatkan berdasarkan rekomendasi Tim RHA untuk memberikan pelayanan kesehatan dengan peralatan yang lebih memadai, minimal terdiri dari:
a) dokter umum dan spesialis
b) apoteker dan tenaga teknis kefarmasian
c) perawat
d) perawat Mahir
e) bidan
f) sanitarian
g) ahli gizi
h) tenaga surveilans
i) entomolog

Pelayanan kesehatan pada saat bencana bertujuan untuk menyelamatkan nyawa, mencegah atau mengurangi kecacatan dengan memberikan pelayanan yang terbaik bagi kepentingan korban.

Tindakan keselamatan diterapkan untuk memberi perlindungan kepada tim penolong, korban dan masyarakat yang terpapar dari segala risiko yang mungkin terjadi dan dari risiko potensial yang diperkirakan dapat terjadi (meluasnya bencana, material berbahaya, kemacetan lalu lintas, dan lain lain).

Pertolongan pertama

Pertolongan pertama dilakukan oleh para sukarelawan terlatih, petugas pemadam kebakaran, polisi terlatih, SAR, tim medis gawat darurat. Pertolongan pertama dapat diberikan di lokasi bencana (pos medis lapangan), sebelum korban dipindahkan, tempat penampungan sementara (pos medis depan), pada “tempat hijau” di pos medis belakang serta dalam ambulans saat korban dipindahkan ke fasilitas kesehatan.

Pos medis lapangan adalah tempat pertolongan pertama di lokasi bencana, dapat berupa tenda perawatan dan puskesmas. Pemilahan korban (triase) dilakukan di pos medis lapangan dan dikelompokkan sesuai tag (warna) tingkat kegawatdaruratan.

Pos medis depan adalah fasilitas kesehatan terdekat dengan lokasi bencana, dapat berupa rumah sakit atau puskesmas rawat inap. Korban yang membutuhkan stabilisasi segera dan pengawasan intensif dapat dirawat di pos medis depan sebelum di rujuk ke pos medis belakang. Apabila pos medis depan adalah rumah sakit yang memiliki fasilitas lengkap maka pos medis belakang menjadi rujukan sekunder jika jumlah korban melampaui kapasitas pos medis depan.

Pertolongan pertama yang diberikan pada korban di setiap pos dapat berupa kontrol jalan nafas, fungsi pernafasan dan jantung, pengawasan posisi korban, kontrol perdarahan, imobilisasi fraktur, pembalutan dan usaha usaha untuk membuat korban merasa lebih nyaman.

Hal hal penting yang harus diingat apabila korban masih berada di lokasi adalah memindahkan korban sesegera mungkin, membawa korban gawat darurat ke fasilitas kesehatan sambil melakukan usaha pertolongan pertama, seperti mempertahankan jalan napas dan kontrol perdarahan. Resusitasi kardiopulmoner (jantung dan paru) tidak boleh dilakukan di lokasi bencana pada bencana massal karena membutuhkan waktu dan tenaga.

Pos medis belakang didirikan sebagai upaya untuk menurunkan jumlah kematian dengan memberikan perawatan efektif (stabilisasi) terhadap korban secepat mungkin. Upaya stabilisasi korban mencakup intubasi, trakeostomi, pemasangan drain thorax, pemasangan ventilator, penatalaksanaan syok secara medikamentosa, analgesia, pemberian infus, fasiotomi, imobilisasi fraktur, pembalutan luka, pencucian luka bakar. Fungsi pos medis lanjutan ini dapat disingkat menjadi “Three ‘T’ rule” (Tag, Treat, Transfer) atau hukum tiga (label, rawat, evakuasi).

Sebelum evakuasi, petugas kesehatan harus melakukan:
1) Pemeriksaan kondisi dan stabilitas pasien dengan memantau tanda tanda vital;
2) Pemeriksaan peralatan yang melekat pada tubuh pasien seperti infus, pipa ventilator/oksigen, peralatan immobilisasi dan lain lain.

Korban tidak boleh dipindahkan sebelum:
1) Korban berada pada kondisi yang paling stabil;
2) Korban telah disiapkan peralatan yang memadai untuk transportasi;
3) Fasilitas kesehatan penerima telah diinformasikan dan siap menerima korban;
4) Kendaraan yang digunakan dalam kondisi layak pakai.

Mengapa tenaga spesialis, dokter umum, paramedis dan penunjang medis dari luar daerah gempa sangat dibutuhkan?
1. Karena memang tenaga medis, para medis dan penunjang medis setempat mungkin saja turut menjadi korban atau masih "trauma" dengan kejadian luar biasa yang menimpa mereka, mungkin baru beberapa hari kemudian mereka siap bekerja dengan optimal dan itupun tidak mungkin langsung diserahkan banyak kasus.
2. Jumlah korban bencana pasti banyak, penyakit yang terjadi pun mungkin 2-5 kali lipat meningkat dibandingkan kalau tidak ada bencana.
3. Fasilitas dan bahan habis pakai untuk medis mungkin jumlahnya terbatas atau malah rusak, jadi bantuan yang datang biasanya diiringi dengan bantuan alat-alat dan bahan habis pakai, seperti obat-obatan dan pendukung lainnya.

Korban tenggelam yang ditemukan selamat dalam tsunami berisiko terkena pneumonia aspirasi air laut. Penyakit ini berbeda dengan pneumonia biasa meski punya gejala nyaris sama. Pneumonia air laut diawali nyeri dada, sesak napas, dan ada riwayat tenggelam.

"Pneumonia biasa diakibatkan infeksi kuman melalui udara saat pasien batuk atau bersin. Sedangkan

pneumonia aspirasi disebabkan adanya benda asing yang masuk ke dalam jaringan paru. Dalam kasus pneumonia aspirasi air laut maka benda asing adalah air laut yang berisiko merusak jaringan paru dan saluran napas.

Risiko pneumonia bisa ditekan terjadi jika korban segera mengeluarkan air yang masuk ke saluran pernapasan dengan batuk. Namun hal ini terjadi jika korban tersedak air laut dalam jumlah terbatas. Jika jumlahnya terlalu banyak, misal dalam kasus tenggelam, sekadar batuk tidak bisa membersihkan air laut yang masuk saluran pernapasan.

Oleh karena itu, pasien pneumonia aspirasi air laut selain pemberian antibiotk sebaiknya menjalani bronkoskopi untuk membersihkan saluran pernapasan (bronchial washing). Dibilas dengan cairan steril berulang kali untuk menjamin kebersihan saluran napas. Bronkoskopi juga untuk mengambil sampel mikroorganisme yang mungkin masuk bersamaan dengan air laut.
Oleh karena itu, tenaga kesehatan yang menangani korban bencana lekas tanggap bila ada pasien dengan riwayat tenggelam (tsunami). Pasien bisa langsung dirujuk ke rumah sakit dengan fasilitas lebih baik untuk penanganan lanjut.

Tim kedaruratan medik respirasi terdiri dari
- tim yang akan bergabung dengan tim RHA untuk evaluasi awal di lokasi bencana
- dan tim bantuan kesehatan yang akan berada pada posisi di Pos Medis Depan (Rumah Sakit terdekat lokasi bencana) dan di Pos Medis Belakang (RS rujukan).

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PULMONARY INFECTION AFTER DISASTERS : HOW TO CHOOSE ANTIBIOTICS

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ABSTRACT

Natural disaster is an inevitable condition that can occur in any region, including Indonesia due to its geographical location and high density population. Natural disaster events, such as flood, earthquake, wildfire, and tsunami are associated with high morbidity and mortality.

Recent findings show that respiratory infections are a major cause of illness and death among victim population. Management of pulmonary infectious patients in disaster areas are challenging, specifically in prescribing appropriate antibiotic. This obstacle comes from numerous factors such as destruction of health facilities, inadequacy of diagnostic tools, lack of medication or antibiotic stocks, or unusual pathogen causes of infection. In this article, we review pulmonary infection effects of natural disasters and how to choose effective antibiotics in disaster setting.

Keywords : antibiotics, disaster, infection, pulmonary

Pendahuluan

Indonesia merupakan negara dengan banyak potensi bencana. Secara geografis, terdapat pertemuan tiga lempeng tektonik besar di Indonesia, yaitu lempeng Indo-Ausralia, Eurasia, dan Pasifik. Aktivitas tektonik yang berkumpul menyebabkan terbentuknya deretan gunung api di sepanjang pulau di Indonesia, yang termasuk dalam *Ring of Fire* atau deret sirkum pasifik. Patahan aktif pada zona ini sering menimbulkan bencana alam, seperti gempa bumi dan tsunami. Pengaruh perubahan iklim, aktivitas manusia yang memperburuk lingkungan seperti perambahan hutan juga berperan dalam meningkatkan risiko bencana hidrometeorogi seperti banjir dan kebakaran hutan. Menurut data dari Badan Nasional Penanggulangan Bencana (BNPB) terdapat 1.829 kejadian bencana alam di Indonesia pada bulan Januari hingga Juli 2019.[1,2]

Pada kejadian bencana, korban tidak hanya masyarakat yang meninggal ataupun terluka, namun juga korban yang sulit mendapatkan akses ke pelayanan kesehatan. Seluruh korban jiwa yang selamat selama bencana memiliki risiko untuk terkena penyakit, termasuk penyakit paru. Kejadian ini bisa terjadi karena inhalasi air atau zat kimia saat tsunami dan banjir. Kepadatan penduduk serta kurangnya higienitas pada area pengungsian juga menimbulkan kerentanan terhadap infeksi paru. Oleh karena itu, penting bagi tenaga kesehatan untuk mengidentifikasi risiko infeksi tersebut dan memahami penanganan yang tepat untuk mengurangi mortalitas dan meningkatkan kualitas hidup korban bencana alam.[3]

Bencana dan Infeksi Paru

A. Tsunami : Tsunami Lung (pneumonia aspirasi)

Tsunami merupakan salah satu ancaman bencana yang terutama menimpa wilayah pesisir Indonesia. Bencana ini umumnya dipicu oleh gempa bumi di laut yang menyebabkan pergeseran vertikal di dasar laut. Tsunami juga dapat dipicu oleh letusan gunung api aktif. Pada tahun 2018 terjadi tsunami di daerah lampung yang menimbulkan 430 korban meninggal dan hilang, serta gempa bumi dan tsunami di Sulawesi Tengah yang menimbulkan 3.324 korban meninggal. Peningkatan kadar air yang tiba-tiba saat tsunami menyebabkan kejadian tenggelam, inhalasi, dan trauma. Aspirasi yang terjadi tidak hanya air, namun juga

zat kimia dari kerusakan infrastruktur akibat tsunami menyebabkan infeksi, hilangnya surfaktan alveolar, edema pulmonar, dan *acute respiratory distress syndrome* (ARDS). Pneumotoraks dan pneumomediastinum juga dapat terjadi terutama sebagai komplikasi pasien yang membutuhkan bantuan ventilator. [3]

Gejala klinis pasien dapat berupa demam yang fluktuatif dan kronik, batuk tidak produktif, sesak, dan dapat bersifat akut maupun subakut (hingga satu bulan) setelah aspirasi atau imersi. Hasil pemeriksaan penunjang dapat berupa konsolidasi, kavitasi,atau infiltrat pada rontgen toraks.[4]

Inoue dkk dalam laporan tiga kasus yang ditemui pada kejadian Tsunami Jepang tahun 2011 mendefinisikan kombinasi pneumonia karena bahan kimia dan bakteri sebagai *tsunami lung*. Zat kimia yang dapat terinhalasi diantaranya berupa minyak, oli, tanah, debu, pasir, dan limbah Aspirasi air laut yang kotor juga meningkatkan risiko pneumonia infektif.[5] Patogen yang dapat ditemukan dari hasil kultur spesimen pada kasus tsunami lung diantaranya adalah *Aeromonas sp.* dan *Burholderia pseudomallei* pada kasus tenggelam air tawar, Francisella philomiragia pada kasus tenggelam air laut, *Pseudomonas aeroginosa* dan *Pseudallescheria boydii* pada tenggelam air terpolusi. Translokasi kolonisasi orofaring oleh bakteri gram positif seperti Streptococcus pneumoniae, Staphylococcus aureus and anaerobes menuju paru juga dapat menyebabkan aspirasi yang menimbulkan pneumonia. *Legionella pneumonia* dan Aspergillosis dapat juga ditemukan pada *tsunami lung.* [3, 5,6]

B. Banjir

Banjir merupakan salah satu bencana hidrometeorogi yang paling sering terjadi di Indonesia. Pada tahun 2018, banjir merupakan bencana kedua terbanyak dengan 871 kejadian.[1,2] Risiko infeksi paru juga meningkat pada banjir. Seperti tsunami, kenaikan level air yang tiba-tiba pada banjir juga menyebabkan aspirasi dan *near drowning*.[3] Selain itu, faktor kerusakan infrastrukrur, kurangnya sanitasi, dan paparan air banjir dan hujan juga meningkatkan risiko infeksi paru. Pada banjir besar Bangladesh tahun 1988, 17,4% dari seluruh penyakit adalah infeksi saluran respirasi dan 13% korban diantaranya meninggal.[7] Kejadian banjir di Pakistan tahun 2010 juga menunjukkan 21% dari total penyakit infeksi adalah respirasi.8 Penelitian kohort yang dilakukan di Bangladesh tahun 2001-2007 oleh Milojevic dkk menunjukkan terdapat peningkatan risiko infeksi respirasi akut pada enam bulan setelah banjir (RR=1.25; 95% ci 1.06-1.47).[9] Terdapat juga laporan peningkatan kejadian tuberkulosis pada korban penyintas banjir di India pada tahun 2008.[10] Infeksi paru karena banjir biasanya disebabkan oleh poli mikroba. Gejala yang timbul dapat berupa batuk kronik, demam, sesak. Komplikasi yang dapat terjadi diantaranya adalah nekrosis paru, terbentuknya abses, dan juga empiema. Penyakit paru lainnya yang dapat terjadi adalah flu viral, COPD, asthma,dan bronkitis alergi.[10]

C. Gempa Bumi

Pneumonia dan Bronkitis

Pada kejadian gempa dan tsunami Aceh 2004, tercatat 37.492 kasus ISPA. Insiden tertinggi terjadi dalam 6 minggu awal pasca bencana. Mulai dari minggu ke-empat, proporsi infeksi saluran napas bawah menurun tajam, dan infeksi saluran napas atas meningkat kembali, menunjukkan terjadinya transisi penyakit kembali ke pola normal.[11]

Bencana gempa bumi yang terjadi di Tohoku, Jepang, juga menyebabkan peningkatan pesat hospitalisasi akibat pneumonia. Dalam kurun waktu 3,5 bulan, insiden pneumonia per minggu meningkat 5,7 kali (IK95%=3,9-8,4) dari *baseline*.[12] Kematian pada pneumonia meningkat 8,9 kali (KI95%=44-17,8) dan rasio laju mortalitas *(mortality rate ratio)* tertinggi terjadi pada pasien yang tinggal di panti wreda.[12] Risiko ISPA

meningkat seiring dengan padatnya pengungsian, buruknya ventilasi udara, dan nutrisi yang kurang.[13]

Pneumonia yang dapat terjadi pada korban gempa bumi sebagian besar merupakan *community-acquired pneumonia.* Gejala yang muncul berupa demam, menggigil, sesak napas, nyeri dada pleuritik, dan batuk dengan sputum purulen. Diagnosis mikrobiologi biasanya sulit dilakukan, apalagi jika pasien tidak terdapat sputum. Selain pneumonia, bronkitis dapat terjadi pada korban gempa bumi. Pasien bronkitis akut menunjukan gejala batuk akut dan non produktif pada pasien yang diketahui tidak memiliki penyakit paru yang mendasari. Pada infeksi bakteri, awalnya akan muncul demam ringan dan menggigil serta sputum yang berubah purulen. Penyebab bronkitis biasanya influenza, rhinovirus. Peran H. influenza, Streptococcus pneumonia masih belum dapat dipastikan dalam infeksi bronkitis akut.[14]

Koksidioidomikosis

Pada kejadian gempa bumi, debu dari bangunan yang rusak dan spora jamur *yang berasal* dari puing bangunan dapat menyebabkan infeksi jamur, yaitu koksidioidomikosis. Infeksi ini disebabkan oleh *Coccidioides immitis* and *C. posadasii*. Wabah koksidioidomikosis pernah terjadi pada Gempa California tahun 1994. Pasien datang dengan keluhan mirip dengan pneumonia komunitas, dengan batuk, nyeri dada pleuritik dan demam. Gejala penyerta dapat berupa nyeri kepala, kelelahan, nyeri otot, ruam, nyeri sendi dan/atau kaku sendi, penurunan kesadaran, kaku leher, atau sensitif terhadap cahaya. Pemeriksaan radiologi dapat menunjukkan gambaran pneumonia. Untuk mengidentifikasi jamur, dapat dilakukan pewarnaan dan kultur, dan akan ditemukan jamur dengan struk sperula. Selain itu, dapat pula dilakukan pemeriksaan serologi.[15,16]

Tuberkulosis

Selain infeksi akut, perlu juga diperhatikan tentang infeksi kronis (seperti tuberkulosis) yang mungkin sudah signfikan sebelum bencana. Selain itu, penularan TB paru juga meningkat pada populasi yang mengungsi karena kerusakan infrastruktur. Penularan TB dapat terjadi akibat sirkulasi udara yang kurang baik, durasi kontak dengan pasien TB, paparan sinar ultraviolet yang rendah dan status gizi yang buruk. Kejadian TB biasanya terjadi pada anak-anak akibat pajanan terhadap penderita TB dewasa. Tingkat penularan TB dan risiko resistensi OAT juga dapat meningkat setelah bencana karena pasien TB tidak mendapatkan program pengobatan, kepatuhan pengobatan yang rendah, resep yang tidak sesuai, pasokan obat yang kurang dan kualitas obat yang buruk.[3]

D. Kebakaran Hutan

Kebakaran hutan menyebabkan terkontaminasinya udara yang dihirup oleh manusia. Berbagai macam komposisi zat beracun akibat kebakaran hutan, diantara karbon monoksida (CO), sulfur dioksida (SO_2), nitrogen dioksida (NO_2), ozone (O3), sianida, partikulat, dan produk pembakaran yang sulit diprediksi, yang dapat menyebabkan asfiksia dan kerusakan pada paru.[15]

Sebuah studi pada kera Rhesus macaque menunjukan bahwa paparan dini terhadap asap dapat mempengaruhi sistem imun, yang diuur dengan berkurangnya sintesis sitokin dalam sel darah perifer, yang bertahan hingga dewasa. Menghirup asap pembakaran kayu jangka pendek dapat membahayakan respon imun paru, dan mungkin inilah yang menjadi alasan kemungkinan meningkatnya infeksi paru pada anak yang terpapar asap kayu.[17]

Pada tahun 1997 terdapat lebih dari 500 kematian akibat kabut asap akibat kebakaran hutan selama tiga bulan di wilayah Indonesia. Pada kejadian yang sama, dilaporkan sekitar 1,5 juta kasus infeksi pernapasan

terjadi.15 Pada kebakaran hutan tahun 2003 di California, tercatat kasus bronkitis akut dan bronkiolitis meningkat 9,6% (IK95%=1,8-17,9%), dan kasus pneumonia pada usia 5-18 tahun meningkat 6,4% (IK95%=2,1-14,2).[18]

Bronkiolitis biasanya terjadi pada bayi dengan gejala hidung berair, batuk, sesak napas, takipnea, mengi dan tanda distres napas. Pemeriksaan radiologi tidak rutin dilakukan. Indikasi pemeriksaan radiologi adalah jika dicurigai terdapat penyakit lain.[19]

Penggunaan Antibiotik pada Penanganan Infeksi

Penggunaan antibiotik yang tidak tepat pada *setting* bencana masih banyak ditemukan. Berdasarkan penelitian yang dilakukan oleh Iwata dkk di kota Ishinomaki pasca bencana gempa dan tsunami Tohoku 2011 menunjukkan hanya 6,9% antibiotik yang diberikan secara tepat, 6,8% alasan pemberian antibiotik dapat diterima, dan 86,3% penggunaan antibiotik tidak tepat.[20]

Umumnya untuk infeksi seperti *common cold,* bronkitis akut, bronkiolitis dan influenza, penggunaan antibiotik tidak direkomendasikan. Meskipun demikian, jika pasien disertai dengan risiko tinggi terjadinya komplikasi, pemberian antibiotik harus segera dilakukan. Pada pasien batuk akut atau bronkitis akut, harus diberikan antibiotik jika disertai dengan pneumonia. Pemberian antibiotik segera dapat dipertimbangkan pada kondisi berikut.[20]

- Pasien dengan sakit berat,
- Pasien memiliki gejala dan tanda sugestif penyakit yang serius dan/atau terdapat komplikasi.
- Pasien dengan risiko tinggi terjadi komplikasi serius akibat adanya komorbid (penyakit jantung, paru, ginjal, hati, neuromuskular, imunosupresi, kistik fibrosis dan bayi prematur)
- Pasien berusia >65 tahun dengan batuk akut dan ≥2 kriteria berikut, atau >80 tahun dengan batuk akut dan ≥1 kriteria berikut:
    - Riwayat rawat inap satu tahun terakhir
    - Diabetes melitus
    - Riwayat gagal jantung kongestif
    - Penggunaan glukokortikoid oral.

Pada kondisi pasien dengan infeksi kronis seperti tuberkulosis, penggunaan antibiotik tetap dilanjutkan dan pemilihan regimen obat disesuaikan dengan program pengobatan anti tuberkulosis yang pasien jalani sebelumnya.

Pneumonia aspirasi.

Pemilihan antibiotik pada kasus *near-drowning-associated pneumonia* merupakan hal yang cukup sulit untuk dilakukan, karena pada banyak kasus terdapat demam dan leukositosis tanpa adanya bukti infeksi. Antibiotik diberikan pada pasien dengan gejala demam, gambaran infiltrat paru dan/atau tanda toksisitas sistemik atau instabilitas hemodinamik tanpa penyebab yang jelas. Meskipun gejala dan tanda tersebut dapat muncul pasca aspirasi tanpa disertai pneumonia, tetap perlu dilakukan pemantauan ketat pada pasien.[22]

Pilihan antibiotik empiris untuk terapi pneumonia aspirasi adalah dengan menggunakan penisilin spektrum luas atau kombinasi dengan penghambat beta laktamase. Jika pasien sakit sedang-berat, perlu dipertimbangkan penambahan aminoglikosida. Klindamisin dan floroquinolon dapat dijadikan alternatif

pada pasien dengan alergi penisilin.[3,22] Pilihan antibiotik definitif dapat diberikan berdasarkan hasil kultur dan hasil tes sensitivias mikroba.

Pada bencana alam, dapat terjadi keterlambatan pasien menuju akses kesehatan dan terdapat keterbatasan pemeriksaan penunjang untuk menganalisa jenis mikroba penyebab infeksi pada pasien. Kondisi tersebut menyebabkan keterlambatan diagnosis dan pasien datang dengan kondisi yang berat. Selain itu, infeksi paru pada tsunami atau banjir disebabkan oleh polimikroba dan risiko resisten terhadap antibiotik yang lazim digunakan sehingga untuk penanganan pasien dapat menggunakan antibiotik spektrum luas. Pada pasien dengan kondisi yang tidak membaik dengan pemberian antibiotik kombinasi, penggunaan antibiotik spektrum yang lebih luas dapat dipertimbangkan, seperti carbapenem, meropenem, atau imipenem.[4,5]

Kesimpulan

Kejadian bencana alam meningkatkan risiko infeksi pada korban, salah satunya infeksi paru. Infeksi tersebut dapat terjadi akibat efek langsung maupun tidak langsung dari bencana.

Pada kondisi bencana, pemberian antibiotik diberikan sesuai dengan diagnosis dan kondisi klinis pasien. Pada pasien dengan risiko komplikasi yang tinggi dan kondisi yang berat, pemberian antibiotik harus segera dilakukan untuk mencegah terjadinya perburukan. Mengenali kondisi bencana dan pengetahuan tentang kemungkinan mikroba penyebab infeksi dapat menjadi dasar pemilihan antibiotik agar penggunaan antibiotik tepat sasaran. Pada kondisi bencana, infeksi polimikroba dan risiko resisten terhadap antibiotik yang rutin diberikan, menyebabkan tenaga kesehatan harus mempertimbangkan dengan cermat pemberian antibiotik baik monoterapi, maupun kombinasi. Pada pasien dengan klinis yang tidak membaik dengan terapi empiris awal, perlu dipertimbangkan antibiotik spektrum yang lebih luas untuk menangani infeksi paru pada pasien.

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EMCO FOR VIRAL PNEUMONIA

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ABSTRACT

Viral pneumonias tend to be mild in adults with the occasional exception of those due to Influenza and Adenovirus. Since SARS in 2003, there has been a proliferation of diagnostic tests allowing us more rapid confirmation of these viral infections. Unfortunately treatment has lagged behind with very few effective anti-viral drugs available for these infections. Sometimes, this leaves us with the critically ill patient a with very severe pneumonia due to viral pneumonias on mechanical ventilation and little else. With the advent of ECMO, there has been spectacular success in a selected group of patients. This discussion touches on the principles of ECMO and how it can be deployed in a highly selected group of patients.

CLASSSIC TO ADVANCE AIRWAY CLEARANCE TECHNIQUE

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ABSTRACT

Introduction

The lungs are remarkably resistant to environmental injury, despite continuous exposure to pathogens, particles, and toxic chemicals in inhaled air. Their resistance depends on a highly effective defense provided by airway mucus, an extracellular gel in which water and mucins (heavily glycosylated proteins) are the most important components. Airway mucus traps inhaled toxins and transports them out of the lungs by means of ciliary beating and cough. [1]

Mucus is produced throughout the bronchial tree by serous cell, goblet or mucus cell, Clara cells, and type II alveolar cells. Mucus is one of the most important lung defence mechanism. The amount of mucus produced at any level in the brochial tree depends on the number of mucus pruducing cells. In normal situasions, without pulmonary diseases, the total amount of mucus that reaches the trachea is about 10 to 100 ml / day. [2]

Effective mucus clearance is essential for lung health, and airway disease is a consistent consequence of poor clearance. Healthy mucus is a gel with low viscosity and elasticity that is easily transported by ciliary action, whereas pathologic mucus has higher viscosity and elasticity and is less easily cleared. The conversion from healthy to pathologic mucus occurs by multiple mechanisms that change its hydration and biochemical constituents; these include abnormal secretion of salt and water, increased production of mucins, infiltration of mucus with inflammatory cells, and heightened bronchovascular permeability. The accumulation of mucus results from some combination of overproduction and decreased clearance, and persistent accumulation can lead to infection and inflammation by providing an environment for microbial growth (Fig. 1). [1]

Patients with airway diseases, such as chronic bronchitis and acute asthmatic episodes, may find symptoms of airway stasis on the airways may contribute to bronchial obstruction. Basically, two mechanisms are responsible for the retention of mucus, hypersecretion and impaire clearance. In cleaning the airway there is also mucus clearance which is actually the mechanism of mucocilary clearance and coughing. [3]

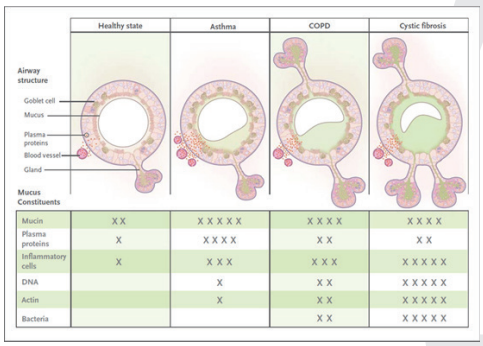


Figure 1. Mucus characteristic and airway mucosal disease[1]

Airway Clearance Techniques

Airway clarance techniques had been started since 1990s. Airway clearance techniques use the gravity to aid mucus transport such as postural drainage (PD). External application of forces against the chest wall are postural drainage, percussion, vibrations/shaking, high-frequency chest wall compression (HFCC). Postural drainage to breathing techniques are known as chest physiotherapy (CPT) along with coughing. [3, 4]

Devices designed to introduce positive pressure and/or oscillation into the airways—positive expiratory pressure (PEP) masks, flutter, cornet, acapella, intrapulmonary percussive ventilation (IPV), and insufflation-exsufflattion. [5]

Studies have shown that a combination of techniques are effective in secretion removal. So, this article discusses the mechanism of airway clearance techniques that will work simultaneously to provide better results.

Postural drainage (PD)

Postural drainage is a therapeutic modality that uses gravity-assisted positioning designed to improve pulmonary hygiene in patients with retained secretions. There are 12 basic positions in which patients can be placed for postural drainage. In fact, positioning in the Trendelenburg and various other positions may acutely worsen hypoxemia. Evidence has shown that a patient's arterial oxygenation may fall during positioning. To accomplish the same goal it is common practice, in intensive care units, to turn patients side to side every 2 hours so as to aid in mobilizing secretions.[4]

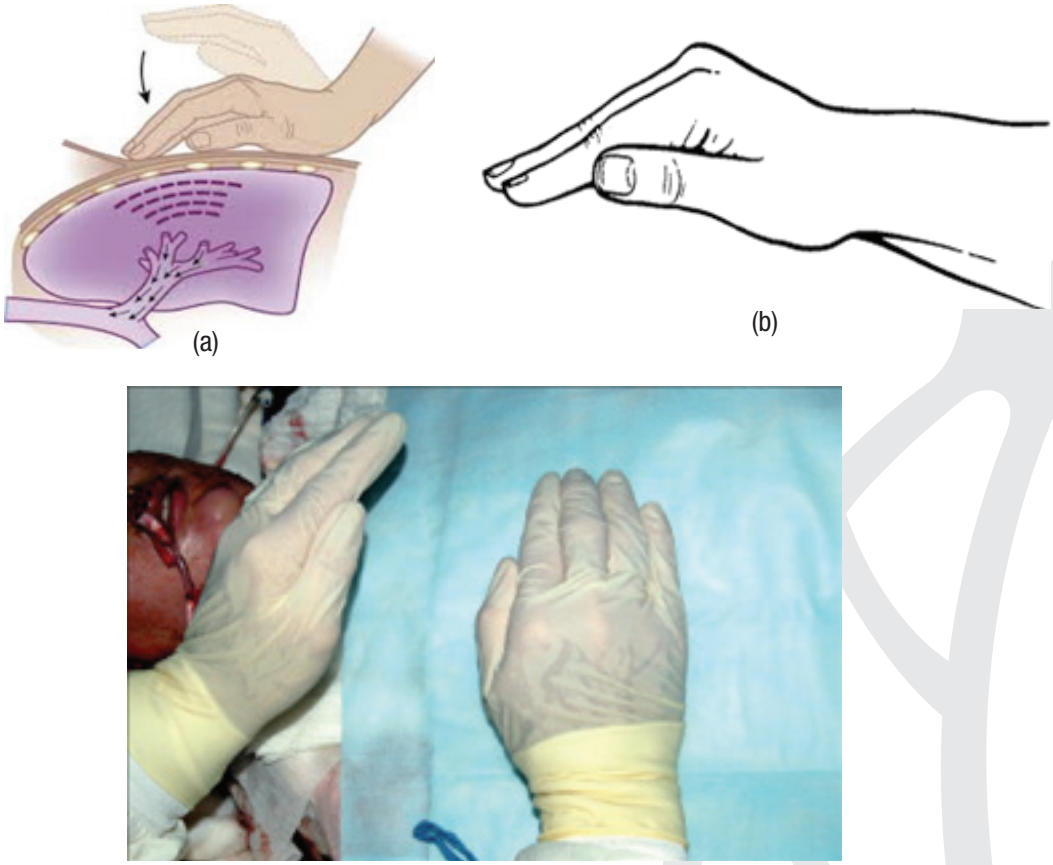
Figure 2. Postural drainage positioning for secretion mobilization[4]

Percussion

Percussion aids in the removal of secretions from the tracheal bronchial tree. Percussion is done by cupping the hand so as to allow a cushion of air to come between the percussor's hand and the patient. If this is done properly, a popping sound will be heard when the patient is percussed. There should be a towel between the patient and the percussor's hand in order to prevent irritation of the skin.[8] Percussion is applied over the surface landmarks of the bronchial segments that are being drained. The hands rhythmically and alternately strike the chest wall. Incisions, skin grafts, and bony prominences should be avoided during percussion . [4]

Percussion depends on the frequency. Research by Jirakit et al with twenty six healthy volunteers (13 women and 13 men), with a mean age of 20.15 ± 1.17 years, participated. They were randomized into three standard positions of PD (upper, middle, or lower lobes) and given manual percussion at a frequency of 240 times per minute for 5 min. [6]

The lung volumes showed no statistical difference in vital capacity (VC) or inspiratory reserve volume (IRV) from percussion during PD in all positions, except for the lower lobe, where increased tidal volume (TV) and decreased ekspiratory reserve volume (ERV) were found when compared to PD alone. Furthermore, percussion during PD of the upper and middle lobes did not affect respiratory rate (RR) or minute ventilation (VE), when compared to PD alone. In addition, percussion during PD of the middle and lower lobes increased VO2 and VCO2 significantly, when compared to PD alone, but it did not influence PD of the upper lobe. [6]



(a) (b) (c)

Figure 3. (a), (b), and (c) are Percussion technique[6]

Vibration/shaking

Chest vibration and chest shaking are manual techniques used to assist in airway clearance. Vibration/ shaking is a shaking movement used to move loosened secretions to larger airways so that they can be

coughed up or removed by suctioning. Vibration involves rapid shaking of the chest wall during exhalation. The percussor vibrates the thoracic cage by placing both hands over the percussed areas and vibrating into the patient, isometrically contracting or tensing the muscles of their arms and shoulders. Mechanical vibrations have been reported to produce good clinical results. Gentle mechanical vibration may be indicated for patients who cannot tolerate manual percussion. Chest physiotherapy techniques should be used every 2–4 hours for patients with retained secretions. Therapy should continue until breath sounds improve.[4]

Research by Li and Silva at 2008, investigate the frequency and force of chest vibration as applied by 18 physiotherapists (5 men and 13 women) working in a teaching hospital, showed that a mean frequency of 5.7, 5.3, and 5 Hz and a mean maximum force of 272.78, 273.47, and 271.13 N for conditions 1 (directly on the chest), 2 (on the chest through a layer of sheet), and 3 (on the chest through a layer of towelling) respectively. [7]

A study investigating vibration on a human model reported a mean frequency of 5.5 Hz and a mean peak force of 137.1 N. [8]

Figure 4. Mechanical chest vibration

Active cycle of breathing technique

The Active Cycle of Breathing Techniques (ACBT) is an active breathing technique performed by the patient and can be used to mobilise and clear excess pulmonary secretions and to generally improve lung function. It is a flexible method of treatment which can be used in conjunction with positioning and adapted for use with most patients. Each component can be used individually or as part of the ACBT cycle depending on the patient's problem. Once ACBT has been taught, the patient can be encouraged to use it independently. [4]

ACBT used to, (1) Loosen and clear secretions from the lungs (2) Improve ventilation in the lung (3) Improve the effectiveness of a cough ACBT consists of three main phases: (1) Breathing Control (2) Deep Breathing Exercises or Thoracic Expansion Exercises (3) Huffing or Forced Expiratory Technique (FET). Additionally, a

manual technique (MT) or positive pressure can be added if and when indicated, to create a more complex cycle to help improve removal of secretions on the lungs. this may include percussion or expiratory vibrations. [4]

Positive Expiratory Pressure (PEP)

Acapella

The Acapella® is a small hand-held device, which combines PEP and high-frequency oscillation therapy. The manufacturers suggest that Acapella® may offer a beneficial alternative to other airway clearance techniques (ACTs) as it requires less therapist time, is self performed, can be used in any postural drainage position and is suitable to patients with a wide range of pulmonary function. No clinical trials have investigated the efficacy of Acapella® during a pulmonary exacerbation requiring oral antibiotic therapy. [9,10]

The Acapella consists of a counterweighted plug and metal strip attached to a lever, and a magnet. Airflow oscillation is created by breaking and reforming of a magnetic attraction by the plug as it intermittently occludes air passing through the device 7. The device is available either in blue, for patients who cannot maintain their expiratory flow above 15 l.min–1 for 3 s, or green, for patients who can maintain an expiratory flow equal to or above 15 l.min^{-1} for at least 3 s .[11]

Figure 5. Acapella device [11]

Flutter

Flutter® is a simple handheld device that allows removal of mucus from the airways using positive expiratory pressure. Flutter® device represents an alternative to traditional physiotherapeutic modalities and has been increasingly used in the management of respiratory conditions characterized by chronic sputum production. [12]

The positive expiratory pressure was shown to be more effective than Flutter® in terms of preserving pulmonary function, hospital admissions, and antibiotic use in patients, who were followed up for 1 year. Daily use of the Flutter® device at home was as effective as ACBT in patients with non-CF bronchiectasis, and it leads to higher levels of adherence by patients. [12]

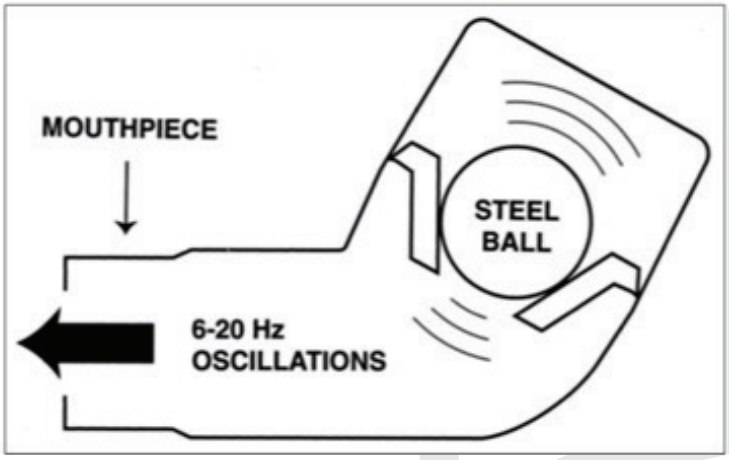


Figure 6. Flutter Device[12]

Insuffaltion Exsufflation
Manually assisted coughing
Air Stacking

Air stacking (AS) is a lung insufflation method that requires the use of a manual insufflator to provide air volumes higher than inspiratory capacity. A manual was used to perform the AS technique. The sum of the inspired volume (IC) and the volume applied by AS, reaching the maximum insufflation capacity (MIC) level, was represented as IC and and AS. IC and AS was achieved with the subject taking a deep breath, starting from functional residual capacity and ending at total lung capacity (TLC), holding it and air stacking consecutive delivered volumes of air until the maximum volume could be held with the glottis closed. Each volume delivered by the manual insufflator was followed by a deep inspiration and sub- sequent closure of the glottis. During this moment, the subjects were instructed to not exhale, holding the amount of air in their lungs. At the end of the set of maneuvers, the mask was removed, and the subject was asked to perform a strong cough starting from IC and AS volume. The highest cough peak flow of 3 attempts was taken into account for analysis and compared with pre- and post-spontaneous coughs. [13]

Assisted cough peak flow (CPF) can be greatly increased in patients receiving maximal insufflations followed by manual thrusts for assisted coughing. In Sarmento et al research, they evaluated 364 neuro muscular disease (NMD) patients able to air stack, the mean vital capacity (VC) in the sitting position was 996.9 mL, the mean MIC was 1647.6 mL, and although CPFs were 2.3 L/s (<2.7 L/s or the minimum needed to eliminate airway secretions) mean assisted CPF were 3.9 L/s. [14]

Techniques of manually assisted coughing involve different hand and arm placements for expiratory cycle thrusts. An epigastric thrust with one hand, while applying counterpressure across the chest to avoid paradoxical chest expansion with the other arm, further increases assisted CPF for 20% of patients . Manually assisted coughing is usually ineffective in the presence of severe scoliosis because of a combination of restricted lung capacity and the inability to effect diaphragm movement by abdominal thrusting because of severe rib-cage and diaphragm deformity. Abdominal compressions should not be used for 1 to 1.5 hours following a meal. When inadequate, and especially when inadequacy is due to difficulty air stacking,

the most effective alternative for generating optimal CPF and clearing airway secretions is the use of mechanical insufflation-exsufflation (MI-E). [14]

The inability to generate more than 2.7 L/s or 160 L/m of assisted CPF despite having a VC or MIC greater than 1 L usually indicates fixed upper-airway obstruction or severe bulbar muscle weakness and hypopharyngeal col- lapse during coughing attempts. Vocal cord adhesions or paralysis may have resulted from a previous translaryngeal intubation or tracheostomy . Because some lesions, especially the presence of obstructing granulation tissue, can be corrected surgically, laryngoscopic examination is warranted especially before decanulation. [14]

Mechanical insufflation-exsufflation

MI-Es deliver deep insufflations followed immediately by deep exsufflations. The insufflation and exsufflation pressures and delivery times are independently adjustable. Insufflation to exsufflation pressures of +40 to –40 cm H_2O are usually the most effective and preferred by most patients. Onset of insufflation generates an insufflation flow peak and a lung insufflation of more than 2 L. Mechanical exsufflation generates two exsufflation flow notches. One occurs when the insufflation pressure stops and is due to the elastic recoil of the lung. The second one, a bit greater, is caused by the exsufflation pressure itself. [15]

MI-E can be provided via an oral-nasal mask, a simple mouthpiece, or via a trans laryngeal or tracheostomy tube. MI-E can be provided via an oral-nasal mask, a simple mouthpiece, or via a trans laryngeal or tracheostomy tube. [15]

The Cough Assist can be manually or automatically cycled. Manual cycling facilitates caregiver-patient coordination of inspiration and expiration with insufflation and exsufflation, but it requires hands to deliver an abdominal thrust, to hold the mask on the patient, and to cycle the machine. [15]

One treatment consists of about five cycles of MI-E or MAC followed by a short period of normal breathing or ventilator use to avoid hyperventilation. Insufflation and exsufflation pressures are almost always from +35 to +60 cm H_2O to –35 to –60 cm H_2O. Most patients use 35 to 45 cm H_2O pressures for insufflations and exsufflations. In experimental models, +40 to –40 cm H_2O pressures have been shown to provide maximum forced deflation VCs and flows. [15]

Conclusion

There are some methods to do airway clearance. They can be done independently, with partner or using tools. Those methods and techniques for carrying out airway clearance should be used simultaneously to get better results in secretion removal.

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PENETRATING CARDIAC INJURY IN SURABAYA

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ABSTRACT

Background: Heart trauma and great vessels are leading causes of death in young adults. The prehospital mortality rate for penetrating cardiac injury was 70 – 80%. Penetrating cardiac injury has the highest morbidity and mortality rates of all organ injury.

Purpose: To show own experience in treatment of patient with penetrating injury to the cardiac.
Material and Method:We conducted a retrospective review of 59 patient with penetrating cardiac injury operated from 1985-2017 (32 years) at 3 hospitals in Surabaya.

Result: There are 59 patients penetrating cardiac injury with male 48 patients (81.35 %) and female 11 patients (18.65 %). Age is ranged from 9-64 years. The mechanism of injury consist: segment fracture of the rib one patient (1.69 %), CVP catheter one patient (1.69), air rifle (bullet) 5 patients (8.47 %), and stab wound (knife, stiletto) 52 patients (88.15 %). The injured site are Pulmonary Artery 1 patients (1.69%), Ascending Aorta one patients (1.69 %), Right Atrium 4 patients (6.78 %), Left Ventricle 5 patients (8.47 %), and Right Ventricle 48 patients (81.37 %).There were 5 major complications: icterus in 14 patients (23.73 %), wound dehiscent in one patient (1.69 %), sepsis in one patient (1.69 %), VSD in one patient (1.69 %), mortality in 5 patients (8.47 %). The surviving is 54 patients (91.53%).

Conclusion : The result of this experience were good in all cases. Because of that, we recommend aggressive thoracotomy exploration for penetrating cardiac injury to lower mortality and morbidity.

Keywords: *Cardiac Injury, Penetrating Trauma*

Notes:
[1]President of Indonesian Association of Thoracic, Cardiac, and Vascular Surgeons *Presented at International Meeting on Respiratory Care Indonesia (Respina) –Jakarta 2019

THE STRATEGY OF PULMONARY INFECTION PREVENTION, DETECTION & TREATMENT IN DISASTER: EARLY MANAGEMENT FOR PATIENTS IN VOLCANIC AREA

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ABSTRACT

Natural disasters are associated with much mortality and morbidity. The World Health Organization defines a disaster as a disruption of society resulting in widespread human, material, or environmental loss that exceeds the affected society's ability to cope by using local resources . Natural disasters can be broadly classified into 3 groups: geophysical (e.g., earthquakes, volcanic eruptions, and tsunamis), hydrometeorological (e.g., floods, hurricanes, and tornadoes), and geomorphological (e.g., landslides and avalanches). New research published in 2011 has thrown light on the significant number of lung diseases in natural disasters. Volcanoes and storms are known to increase harmful suspended particles like volcanic ash and toxic gas in air, not only at the time of the event but also for a long time there after. People with pre existing diseases like COPD are more affected, but healthy subjects may also develop acute symptoms like bronchospasm and hemoptysis. Infectious diseases, both common ones like influenza and rare ones like Nocardia, occur with increasing frequency. Respiratory pathologies are often neglected in triage protocols. However, proper care for respiratory diseases can significantly decrease mortality.

Infectious disease outbreaks after natural disasters are uncommon. However, features of the post-impact and recovery phases of disasters, such as population displacement, low vaccine coverage for vaccine-preventable diseases, inadequate sanitation and hygiene infrastructure, and limited access to health care services, can interact to increase the risk for transmission of infectious diseases that were previously established in the affected area

In volcanic eruptions, we get smog: sulfur dioxide and other gases released from the volcano and their reaction products with atmospheric elements. By far, the most harmful elements are the sulfur compounds. Also there is fine volcanic ash suspended in air for a long time. These suspended particles are small enough to enter lower airways. Also, most of these particles are acidic, that can directly irritate the mucosa. Thus existing chronic lung diseases like COPD and asthma are exacerbated and also, the sulfur compounds can impair the local immune system of the lung.

People living near a volcano can present with acute respiratory emergencies like dyspnea and hemoptysis. Postmortem studies on victims killed by the 1982 St. Helens volcanic eruption demonstrated that over eighty percent died as a result of asphyxiation due to bronchial obstruction following ash inhalation. Potential respiratory symptoms from the inhalation of volcanic ash depend on a number of factors, including airborne concentration of total suspended particles, proportion of respirable particles in the ash (less than 10 microns in diameter), frequency and duration of exposure, presence of free crystalline silica and volcanic gases or aerosols mixed with the ash, meteorological conditions, and host factors (existing health conditions and the propensity of those exposed to incur respiratory problems), and the use of respiratory protective equipment. Now, a new term called **Pneumonoultramicroscopicsilicovolcanoconiosis** has been coined to describe the lung problem in volcanic eruptions. This is shortened as **P45.** This is now the longest word in English Dictionary.

Risk for Lung Infection:
Ash inhalation increases risk of lung infection. Inhaling volcanic ash could weaken the body's natural defenses against infection, a recent study concludes. A team of researchers collected samples of ash from the 2010 Eyjafjallajökull volcano eruption in Iceland and, in laboratory tests, found that they reduced the ability of immune cells in lungs to fight off bacterial infections. The ash was found to affect immune cells in the air sacs called 'macrophages'. One important role of macrophages is to identify and kill disease agents, such as bacteria, by ingesting or 'eating' them. In the tests, the researchers watched the cells taking up the ash and found it interfered with their ability to carry out their digestion processes. Macrophages consumed similar numbers of bacteria whether or not they were exposed to ash, but those exposed to ash did not kill as many. Ash also increased the growth of P. aeruginosa bacteria. The researchers conclude that while the effects of ash on lung cells are minimal, exposure to ash could influence respiratory health by damaging the body's immune response to disease and allowing harmful bacteria to multiply.

Major health threats from a volcanic eruption
Volcanoes spew hot, dangerous gases, ash, lava, and rock that are powerfully destructive. People have died from volcanic blasts. The most common cause of death from a volcano is suffocation. Volcanic eruptions can result in additional threats to health, such as floods, mudslides, power outages, drinking water contamination, and wildfires. Health concerns after a volcanic eruption include infectious disease, respiratory illness, burns, injuries from falls, and vehicle accidents related to the slippery, hazy conditions caused by ash. When warnings are heeded, the chances of adverse health effects from a volcanic eruption are very low.

Respiratory effects:
In some eruptions, ash particles can be so fine that they are breathed deep into the lungs. With high exposure, even healthy individuals will experience chest discomfort with increased coughing and irritation. Common acute (short-term) symptoms include:

- Nasal irritation and discharge (runny nose).
- Throat irritation and sore throat, sometimes accompanied by dry coughing.
- People with pre-existing chest complaints may develop severe bronchitic symptoms which last some days beyond exposure to ash (for example, hacking cough, production of sputum, wheezing, or shortness of breath).
- Airway irritation for people with asthma or bronchitis; common complaints of people with asthma include shortness of breath, wheezing and coughing.
- Breathing becomes uncomfortable.

In rare circumstances, long-term exposure to fine volcanic ash may lead to serious lung diseases. For these diseases to occur, the ash must be very fine, contain crystalline silica (for the disease silicosis to occur) and the people must be exposed to the ash in high concentrations over many years. Exposure to crystalline silica in volcanic ash is typically of short duration (days to weeks), and studies suggest that the recommended exposure limits (similar in most countries) can be exceeded for short periods of time for the general population. People suffering from asthma or other lung problems such as bronchitis and emphysema, and severe heart problems are most at risk.

Volcanic ash
Exposure to ash can be harmful. Infants, elderly people, and people with respiratory conditions such as asthma, emphysema, and other chronic lung diseases may have problems if they breathe in volcanic

ash. Ash is gritty, abrasive, sometimes corrosive, and always unpleasant. Small ash particles can abrade (scratch) the front of the eye. Ash particles may contain crystalline silica, a material that causes a respiratory disease called silicosis.

The importance of grain size
Penetration of ash particles into the respiratory tract is largely dependent on particle size. Larger particles (10-100 μm diameter) lodge in the upper airways, while those in the 4-10 μm size range deposit in the trachea and bronchial tubes. Very fine (< 4 μm diameter) particles may penetrate deeper into the lungs, into the alveolar region.
Deposition of relatively coarse particles in the upper airways is primarily associated with symptoms such as irritation of the nose and throat. Deposition of smaller particles in the thoracic region (bronchial tubes and bronchioles) is thought to be associated with acute disease outcomes such as exacerbation of asthma and bronchitis. Very fine particles are termed 'respirable' and may penetrate into the deep lungs where chronic, particle-related respiratory diseases, such as silicosis, are activated.

Crystalline silica
The most hazardous eruptions are those generating fine-grained ash with a high content of free crystalline silica, as this mineral has the potential to cause silicosis (a chronic lung disease resulting in scarring damage to the lungs and impairment of their function). Silicosis is primarily an occupational disease associated with occupations such as stone-cutting, road and building construction and quarrying.
Some volcanoes mass-produce crystalline silica in lavadomes. These are viscous lava piles which grow within volcanic craters and are prone to collapse, generating airborne fine-grained ash rich in free crystalline silica. At Soufrière Hills volcano, Montserrat, West Indies, an eruption began in 1995 and was intermittently active until 2010. This eruption generated dome collapse ash composed of up to 25 wt.% crystalline silica, prompting the UK government to implement controls to minimise population exposure. Comprehensive studies of population exposure to respirable crystalline silica have suggested that the majority of the population are not exposed to sufficiently high airborne concentrations to be at risk of developing silicosis, but a smaller group of individuals (such as outdoor workers) may be at risk of developing mild silicosis.
To date, no longer-term diseases such as silicosis have been attributed to exposure to volcanic ash, although this may be due to inadequate case collection.

Gases
Most gases from a volcano quickly blow away. However, heavy gases such as carbon dioxide and hydrogen sulfide can collect in low-lying areas. The most common volcanic gas is water vapor, followed by carbon dioxide and sulfur dioxide. Sulfur dioxide can cause breathing problems in both healthy people and people with asthma and other respiratory problems. Other volcanic gases include hydrogen chloride, carbon monoxide, and hydrogen fluoride. Amounts of these gases vary widely from one volcanic eruption to the next.

Although gases usually blow away rapidly, it is possible that people who are close to the volcano or who are in the low-lying areas downwind may be exposed to levels that may affect health. At low levels, gases can irritate the eyes, nose, and throat. At higher levels, gases can cause rapid breathing, headache, dizziness, swelling and spasm of the throat, and suffocation.

THE INDIRECT EFFECTS OF NATURAL DISASTERS ON THE LUNG

Communicable respiratory diseases

Communicable diseases, including those of the lung, commonly emerge following disasters. This occurs due to population displacement, poor availability of safe water and sanitation facilities, overcrowding and the non-functional state of health-care services in affected areas. These are often compounded by a poor underlying health and low vaccination status of the affected population. Overcrowding is a common problem in populations displaced by natural disaster and may facilitate the transmission of communicable diseases, particularly respiratory and gastrointestinal diseases. In addition, the lack of access to health services and to antimicrobial agents for treatment increases the risk for death from acute respiratory infection (ARI) following disasters.

The ARI due to *viral diseases* in crowded refugee/displaced population settings spreads quickly. Overcrowding is an important pulmonary risk for healthy displaced survivors , especially children and also for those injured and for those caring for them in makeshift hospitals because of the ease of spread of organisms .This can be prevented by reducing the concentration of infectious respiratory aerosols in the air and by reducing the presence of contaminated surfaces and items with the following methods:

1. Adequate ventilation to ensure good air flow and prevent increased concentrations of respiratory particles.
2. Separating infected patients from other patients to reduce the risk of transmission of infection from the source patient to others by reducing direct or indirect contact transmission.
3. Limiting contact between infected and uninfected people, such as nonessential health-care workers and visitors, which reduces the risk of transmission to susceptible individuals.
4. Spatial separation (>1 m) between patients including head-to-toe positioning of patient beds if space is limited.
5. Cleaning and disinfection of contaminated surfaces and items.

Rescuers and health workers may also be at increased risk of ARI. For example, after the 2008 Wenchuan, China earthquake, victims and rescuers lived in close proximity in temporary shelters with crowded conditions with poor ventilation. The rate of ARI among the victims and rescuers that lived in these shelters was very high, particularly acute upper respiratory infections.

Transmission of *pulmonary tuberculosis* (TB) is also increased in displaced populations following natural disasters. The transmission of TB is facilitated by recirculation of exhaled air, closeness and duration of contact to persons with TB, low exposure to ultraviolet light and poor nutritional status. This is especially so in children, in whom risk of TB is usually followed by exposure to adult TB.

Transmission rates can also increase following disasters because victims default from TB treatment programmes, particularly if they are part of a mobile refugee population. Thus, it is important to maintain adequate antituberculosis treatment programmes following disasters. Acquired TB drug resistance can also occur post disasters due to poor adherence to treatment, inappropriate prescription, irregular drug supply and poor drug quality. The lessons learned from Hurricane Katrina, New Orleans 2005 provides an example of how to minimize the risk of transmission of TB—the strategic elements included:

(i) supplying 2 weeks or 30 days of medicine to each patient who was likely to relocate;
(ii) providing each patient with a personal card listing contact numbers of the TB programme personnel;

(iii) sending a list of patient names to the National Tuberculosis Control Association for sharing with programme officials in other states; and
(iv) establishing a referral center.

Conclusion:

Pulmonary problems are major causes of morbidity and mortality following natural disasters. It is therefore vital that disaster preparedness and response teams are aware of these problems. Pulmonologists should be a part of the team.

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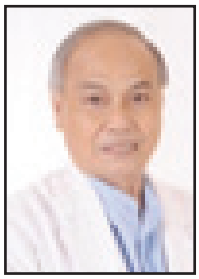
ICU SETTING IN DISASTER AREA



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ABSTRACT

The Task Force for Mass Critical Care Summit Meeting in 2007 and 2014 are discussed with suggestions and recommendations with the hope that each lesson learned will make authorities and leaders more circumspect of the current and present danger.

Definitive Care for the Critically Ill During a Disaster: Current Capabilities and Limitations*

From a Task Force for Mass Critical Care Summit Meeting, January 26–27, 2007, Chicago, IL

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In the twentieth century, rarely have mass casualty events yielded hundreds or thousands of critically ill patients requiring definitive critical care. However, future catastrophic natural disasters, epidemics or pandemics, nuclear device detonations, or large chemical exposures may change usual disaster epidemiology and require a large critical care response. This article reviews the existing state of emergency preparedness for mass critical illness and presents an analysis of limitations to support the suggestions of the Task Force on Mass Casualty Critical Care, which are presented in subsequent articles. Baseline shortages of specialized resources such as critical care staff, medical supplies, and treatment spaces are likely to limit the number of critically ill victims who can receive life-sustaining interventions. The deficiency in critical care surge capacity is exacerbated by lack of a sufficient framework to integrate critical care within the overall institutional response and coordination of critical care across local institutions and broader geographic areas. (CHEST 2008; 133:8S–17S)

Key words: disaster medicine; influenza pandemic; mass casualty; medical care; surge capacity

Abbreviations: DMAT = Disaster Medical Assistance Team; ED = emergency department; NDMS = National Disaster Medical System

Mass casualty events occur frequently worldwide[1]; fortunately, the majority of these do not generate overwhelming numbers of critically ill or injured victims requiring definitive critical care. Mass critical care events, though, have garnered increasing attention[2] and stimulated new interest in critical care disaster preparedness.[3–6] In 2004, an analysis[3] of US critical care disaster response identified major limitations to respond to serious epidemics. In light of the increasing consternation about a potential influenza pandemic,[1,4,5] an updated review of critical care response capabilities is warranted.

Authorities continue to call for development of comprehensive guidance for managing mass casualty events.[2,3] A number of efforts are underway to meet this need, but detailed guidance regarding how to provide critical care for large volumes of patients remains underdeveloped.[5,6] To this end, the Task Force for Mass Casualty Critical Care (hereafter called the *Task Force*) was convened. The Task Force steering committee members (listed in the Appendix) were fairly certain that current critical care surge capacity for disasters had a number of limitations. However, the specific strengths and weaknesses of critical care response capabilities must be delineated to best inform development of novel strategies to augment critical care. This manuscript summarizes the current US and Canadian critical care disaster response capabilities, and provides the rationale and context for the guidance of the Task Force for critical care surge capacity and allocation of scarce life-sustaining interventions.

CHEST

Introduction and Executive Summary

Care of the Critically Ill and Injured During Pandemics and Disasters: CHEST Consensus Statement

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PODCAST

Natural disasters, industrial accidents, terrorism attacks, and pandemics all have the capacity to result in large numbers of critically ill or injured patients. This supplement provides suggestions for all of those involved in a disaster or pandemic with multiple critically ill patients, including front-line clinicians, hospital administrators, professional societies, and public health or government officials. The current Task Force included a total of 100 participants from nine countries, comprised of clinicians and experts from a wide variety of disciplines. Comprehensive literature searches were conducted to identify studies upon which evidence-based recommendations could be made. No studies of sufficient quality were identified. Therefore, the panel developed expert-opinion-based suggestions that are presented in this supplement using a modified Delphi process. The ultimate aim of the supplement is to expand the focus beyond the walls of ICUs to provide recommendations for the management of all critically ill or injured adults and children resulting from a pandemic or disaster wherever that care may be provided. Considerations for the management of critically ill patients include clinical priorities and logistics (supplies, evacuation, and triage) as well as the key enablers (systems planning, business continuity, legal framework, and ethical considerations) that facilitate the provision of this care. The supplement also aims to illustrate how the concepts of mass critical care are integrated across the spectrum of surge events from conventional through contingency to crisis standards of care.

CHEST 2014; 146(4_Suppl):8S-34S

ABBREVIATIONS: CCTL = Critical Care Team Leader; CHEST = American College of Chest Physicians; HC/HA = health-care coalition/regional health authority; IT = information technology; MCC = mass critical care; NGO = nongovernmental organization; Spo_2 = oxygen saturation by pulse oximetry; WHO = World Health Organization

Accepted: May 1, 2014; originally published Online First: August 21, 2014.

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FUNDING/SUPPORT: This publication was supported by the Cooperative Agreement Number HUOTP000391-01 from the Centers of Disease Control and Prevention, and through a research sub award agreement through the Department of Health and Human Services grant Number 1 - HITEP000013-01-00 from the Office of Preparedness of Emergency Operations. In addition, this publication was supported by a grant from the University of California - Davis.

COI grids reflecting the conflicts of interest that were current as of the date of the conference and voting are posted in the online supplementary materials.

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DOI: 10.1378/chest.14-0732

INTERVENTION APPROACH IN MUCOUS CLEARANCE FOLLOWING NATURAL DISASTER

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ABSTRACT

The extensive epithelial surface of the respiratory tract between the nose and the alveoli is exposed daily to viral and bacterial pathogens, particulates, and gaseous material with potentially harmful effects. In response to these challenges, humans have developed a series of defense mechanisms to protect the airways from these insults, thereby maintaining the lungs in a nearly sterile condition. Lung defense involves cough, anatomical barriers, aerodynamic changes, and immune mechanisms; however, the primary defense mechanism is mucociliary clearance (MCC). Healthy airway surfaces are lined by ciliated epithelial cells and covered with an airway surface layer (ASL), which has two components, a mucus layer that entraps inhaled particles and foreign pathogens, and a low viscosity periciliary layer (PCL) that lubricates airway surfaces and facilitates ciliary beating for efficient mucus clearance. The coordinated interaction of these components on the surface of the respiratory tract results in MCC.

The primary symptoms of impaired mucociliary clearance are productive cough and dyspnea. Dyspnea is a result of mucus obstructing airflow in numerous airways, which in cases of total obstruction can lead to atelectasis. Coughing can to some extent substitute for impeded ciliary clearance. This may help explain why diseases in which only the cilia are involved (primary ciliary dyskinesia) tend to be less severe than those primarily caused by dehydration of the mucus (cystic fibrosis) as this will impede both ciliary and cough clearance. Mucociliary clearance is crucial in the pathogenesis of many different diseases, which has led to the development of a wide range of therapies seeking to augment the clearance of mucus from the airways. Roughly, these therapies can be divided into two categories physiotherapeutic regimens and pharmacological therapies.

Many pulmonary complications that occur following natural disasters are a direct result of the disaster itself. The mechanism of insult to the lung as a consequence of a natural disaster will vary depending on the nature of events, but in broad terms can be considered under the following categories:
1) Inhalation of respirable particles, smoke or other toxic gases
2) Aspiration of water and water borne pathogens
3) Direct trauma to the chest
4) Psychological effects causing respiratory symptoms

Direct thermal injury to the upper airways is common but thermal injury to the lower airways is rare. As well as carbonaceous particulates (soot), wood smoke contains a diverse variety of respiratory irritants, including sulphur oxides, nitrogen oxides, phenols, formaldehyde and gaseous acids and alkalis, which cause mucosal inflammation and acute lung injury. Poorly water-soluble substances may cause delayed injury up to 48 h after exposure. In addition, absorption of systemic toxins (carbon monoxide and hydrogen cyanide), generated through incomplete combustion, lead to impairment of oxygen delivery and cellular respiration.

The manifestations of an acute smoke inhalational injury may not occur immediately, and can take several hours to develop. The CXR is an insensitive early indicator of smoke inhalation injury and serial X-rays may be required to detect the development of pulmonary edema/ARDS. In intubated patients, direct inspection of the airways by bronchoscopy if available, allows visualization and assessment of the degree of mucosal injury.

Early intubation should be considered for any patient where a significant inhalational injury is suspected and as such it is important that personnel with appropriate airway management skills are involved at an early stage. Nebulization of adrenaline and corticosteroids has been used to try and minimize airway edema although there is no conclusive evidence of efficacy.

A sudden rise in water levels (a tsunami, hurricane or a flash flood) is more likely to directly lead to drowning, aspiration and traumatic injury than a more gradual or predictable rise. Aspiration of water into the lung can lead to the introduction of infection, loss of alveolar surfactant, pulmonary edema and ARDS. Pulmonary edema is more common in salt water immersion than fresh water. In addition, vomiting of swallowed water can lead to the aspiration of gastric contents, especially if consciousness and airway protective reflexes are impaired. Signs of significant aspiration are usually detectable clinically, such that those with no signs of aspiration on presentation—no coughing, normal examination, normal blood gases and normal CXR—have a very low likelihood of developing pulmonary edema or pneumonia and are unlikely to require further medical intervention.

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ABTRACT FREE PAPER

POLY-PHYTOPHARM IMPROVES ASTHMA CONTROL AND BIOMARKERS (EOSINOPHILS AND miR-126): QUASI-EXPERIMENTAL STUDY IN BRONCHIAL ASTHMA PATIENTS

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ABSTRACT

Background: The level of asthma control is an indicator of asthma management outcomes. It is influenced by a complex immunological mechanism, included miR-126. We aimed to investigate the effect of Poly-Phytopharm on the asthma control tests (ACT) score, blood eosinophils, and serum miR-126 relative expressions in asthma patients. **Methods:** This study used quasi-experimental methods, in 15 stable asthma patients who were not fully controlled at the pulmonary outpatient clinic of Dr. Saiful Anwar General Hospital Malang. Assessment of ACT score, blood eosinophils, and serum miR-126 relative expressions are carried out before and after supplementation of Poly-Phytopharm that consist of Nigella sativa 100 mg, *Kleinhovia hospita* 100 mg, *Curcuma xanthorrhiza* 75 mg, and *Ophiocephalus striatus* 100 mg extract, three times a day, two capsules respectively for 12 weeks. The ACT was scored by the investigator by direct questioning of patients, the blood eosinophil was measured with blood analysis, and the relative expressions of miR-126 in serum was detected with qPCR method. **Result:** There is significant increase of ACT score (18.07 ± 2.57 to 22.06 ± 1.83, p = 0.001). In subject with baseline eosinophils ≥ 300 pg/mL (n = 9), there were significant reduction in blood eosinophils (653.15 ± 276.15 pg/mL to 460.66 ± 202.04 pg/mL, p = 0.038), and an increase of serum miR-126 relative expressions (1.83 ± 1.89 to 5.89 ± 1.34, p = 0.038). **Conclusion:** The administration of Poly-Phytopharm improves asthma control, decreases blood eosinophils, and increases miR-126 relative expressions on not fully controlled stable asthma patients.

Keyword: Poly-Phytopharm, Asthma Control Test, Eosinophil levels, miR-126 relative expressions

BACKGROUND

Asthma is a heterogeneous disease, usually characterized by chronic inflammation of the airways. It is defined by the history of respiratory symptoms that vary over time and in intensity, together with expiratory airflow limitation.[1] The prevalence of asthma is increasing especially in developing countries, which causes an increase in health costs, disability and mortality.[2] The prevalence of asthma in Indonesia in 2013 was 4.5% of the total population based on symptoms.[3]

Achieving controlled asthma is still a significant problem for most patients, despite medications and guidelines for asthma management are widely available. One cause of unsatisfied symptom control is a lack of adherence to the use of inhalers.[4] Besides, even though the patient is obedient and uses devices according to the guidelines, almost 50% of asthma patients have not achieved satisfied symptom control.[1] Many studies on the use of vitamins and additional nutrients in asthma and show varying results. Vitamin D administration has been shown to increase the ACT score in asthma patients who have vitamin D deficiency.[5] Various studies on the use of herbs have been reported; *Nigella sativa* extract supplementation is one of them. The prophylaxis effect of *Nigella sativa* on asthma and bronchitis has been explained in animal and human model studies.[6] *Thymoquinone* contained in Nigella sativa has an immunomodulatory effect. Significantly, thymoquinone can suppress the expression of IL-5 and IL-13 induced by lipopolysaccharide and protein without changes in IL-10 production.[7]

One way to assess the control of asthma symptoms is by using a numeric tool of asthma control; it is the Asthma Control Test (ACT).1 The ACT is straightforward, and this questionnaire is patient-oriented to assess asthma control.[8] In the laboratory, various biomarkers can be used. Blood eosinophils are easily accessible biomarkers.[9] Eosinophils are essential and vital inflammatory cells, marking or inflammatory characteristics in asthma.[10] In evaluating the effectiveness of using mepolizumab, the researchers chose blood eosinophil monitoring as a target for asthma treatment.[11]

Different mechanisms are known to be involved in the process of developing asthma. One of the critical molecules that participated in the regulation of the above process is *microRNA* (miRNA). miRNA can transfer biological information and play an important role in pulmonary inflammatory diseases and allergies.[12] Previous research in animal and human models shows a unique role of miR-126 in the regulation of important features of allergic diseases including asthma.[13] These biomarkers are relatively new and on studied but still very rarely used to evaluate treatment outcomes in asthma patients.

This study aims to prove the effects of Poly-Phytopharm on biomarker levels of inflammation and the score of the asthma control test on not fully controlled stable asthma patients.

MATERIAL AND METHODS

This study uses quasi-experimental design, pre and post treatment on not fully controlled stable asthma patients. Subjects were bronchial asthma patients who were not fully controlled with an age range of 18-59 years. The exclusion criteria were as follows: other lung diseases, malignancy, active smoker, pregnant women, taking any preparation containing Poly-Phytopharm content, known the history of hypersensitivity to Poly-Phytopharm content and taking medications that may interact with Poly-Phytopharm content such as anticoagulant/ antiplatelet, central nervous system depressants and immunosuppressants.[6] Samples were obtained consecutively in asthma patients who met the inclusion and exclusion criteria.

The study was conducted at the Pulmonary Outpatient Clinic and Clinical Pathology Laboratory of Dr. Saiful Anwar Malang, November 2018 - April 2019. Each procedure carried out has been approved by the ethics committee of the General Hospital Dr. Saiful Anwar Malang. Patients volunteered to participate in this study and signed informed consent. The treatment product is produced according to pharmaceutical good manufacturing practice standards by PT. Royal Medicalink Pharmalab and licensed as a Poly-Phytopharm product in Indonesia. Subjects received a dose of two capsules three times for 12 weeks. Every 4 weeks, the patient returned for follow-up, and after 12 weeks, they were assessed for the completion of outcomes. During the treatment period, patients can contact the investigators by phone if there is any appearance of side effects.

ACT Assessment

ACT assessment was carried out at the beginning of the study and after giving treatment for 12 weeks to determine the levels of asthma control and ACT score. The investigators scored the ACT by direct questioning of patients. The ACT consisted of 5 questions with five answer choices, respectively. The lowest value is 5, and the highest value is 25, which is called fully controlled asthma.

Blood Sampling and Biomarkers Measurement

Venous blood samples were collected twice, at the baseline and after 12 weeks of treatment to measure the absolute value of blood eosinophils count and the relative expression of serum miR-126. Three milliliters of blood is used for eosinophil examination through counts of white blood cells with flow cytometry that works

automatically. And two milliliters are used for serum miR-126 relative expression analysis. The miR-126 expression examination consists of serum DNA extraction, cDNA synthesis, and detection of miR-126 using the qPCR method. This procedure was performed using miRNeasy Serum / Plasma Advanced Kit (paint. No. 217204) at the Clinical Pathology Laboratory of Dr. Hospital. Saiful Anwar Malang.

Statistic Analysis

The total sample in our study is 16. The data were recorded in the medical record to then be processed, analyzed and interpreted. The *Shapiro-Wilk* test determaines the normal distribution of data. We use paired t-test to test the effect of Poly-Phytopharm administration to ACT score, blood eosinophil levels, and relative expressions of serum miR-126 on normal data distribution. In the abnormal data distribution, we use the Wilcoxon test. The measurement results for the measured variables were carried out using the Microsoft Office Excel 2010 programs, and statistical analysis using SPSS series 25.0.

RESULTS

A total of 16 stable asthma patients were assessed as meeting the research requirements. One patient did not complete the study because she did not take Poly-Phytopharm regularly as agreed at the beginning of the study. Subsequently, 15 subjects met the requirements for statistical analysis at the end of the study. Socio-demographic characteristics can be seen in table 1. All routine research subjects used asthma control therapy, namely a combination of inhaled corticosteroids and long-acting β2-agonists, but not all of subjects used additional reliever medication (short acting β-agonist, SABA).

Table 1. Sosiodemographic and clinical characteristics

Characteristics	n	%
Age (years)		
∞ 18-40	5	33
∞ 41-59	10	67
Gender		
∞ Male	1	7
∞ Female	14	93
Educational level		
∞ Basic	9	60
∞ Higher	6	40
Occupation		
∞ Employed	7	47
∞ Unemployed	8	53
Atopic in other site(s)		
∞ Yes	14	93
∞ No	1	7

Family history of atopic		
∞ Yes	10	67
∞ No	5	33
SABA usefull		
∞ Yes	9	60
∞ No	6	40
Smoke exposure history		
∞ Yes	9	60
∞ No	6	40
Comorbidity		
∞ Yes	2	13
∞ No	13	87
Blood eosinophil levels		
∞ < 300 pg/mL	6	40
∞ ≥ 300 pg/mL	9	60

Asthma Control Test (ACT)

The primary outcome of our study was the ACT score. Twelve-weeks Poly-Phytopharm administration was proven to improve the ACT score from 18.07 ± 2.57 to 22.06 ± 1.83 and was statistically significant with the p-value = 0.001 as shown in Figure 1. The number of subjects who had an ACT score of less than 20 decreased, which was initially 60%, to only 6.7% after receiving Poly-Phytopharm supplementation.

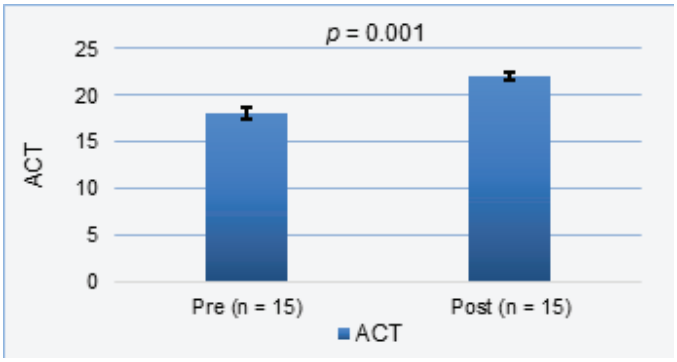

Figure 1. Change of ACT score in both before and after treatment

Blood Eosinophil Levels

We analyzed groups of subjects with blood eosinophil levels at baseline ≥ 300 pg/mL (n = 9). Blood eosinophil levels in the study subjects before treatment were 653.15 ± 276.15 pg/mL, decreasing to 460.66 ± 202.04 pg/mL after the administration of Poly-Phytopharm with p-value = 0.038. It can be concluded that Poly-Phytopharm administration can significantly reduce blood eosinophil levels, as shown in the following figure 3.

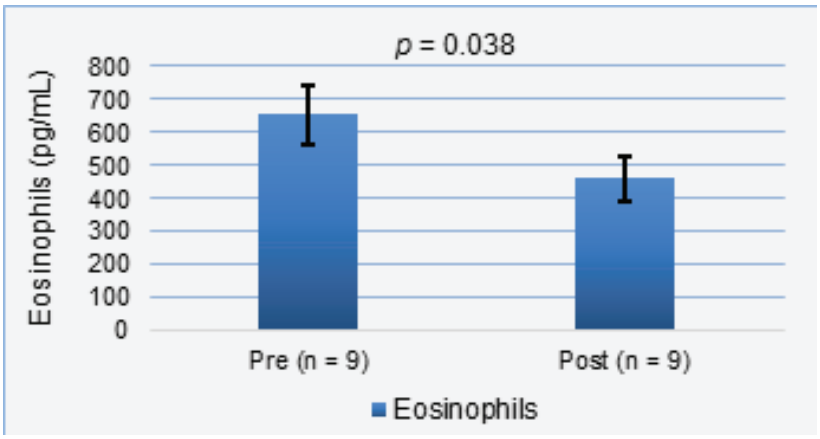

Figure 2. Change of blood eosinophil level in both before and after treatment

Serum miR-126 Relative Expressions

The miR-126 expression is often associated with inflammatory regulation, including eosinophil levels. In this study, we analyzed the relative expression of serum miR-126 in subjects with blood eosinophil levels at baseline ≥ 300 pg/mL (n = 9). We found that the relative expression of serum miR-126 measured before treatment was 1.83 ± 1.89, increasing to 5.89 ± 1.34 after 12 weeks of Poly-Phytopharm administration with p-value = 0.038. Thus, it can be concluded that the administration of Poly-Phytopharm can significantly increase serum miR-126 relative expressions, as shown in figure 3.

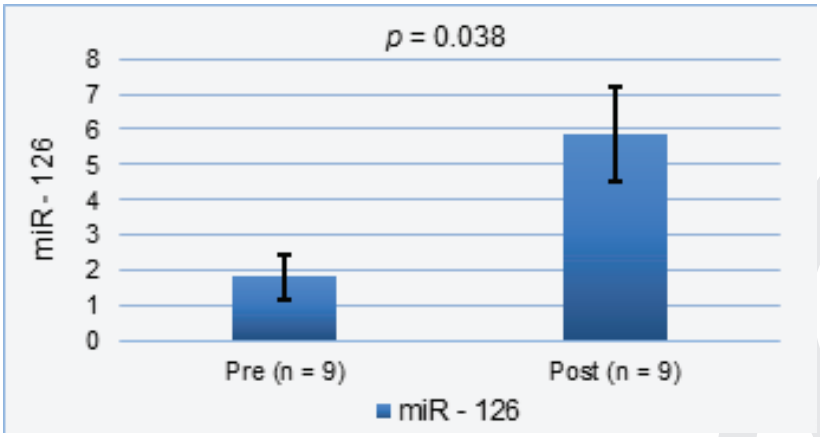

Figure 3. Change of serum miR-126 relative expressions in both before and after treatment

DISCUSSION

Poly-Phytopharm Increases Asthma Control Test (ACT)

In our study, the administration of Poly-Phytopharm can increase the ACT score. The number of subjects who initially had an ACT score of less than 20% was as much as 60%, decreasing to only 6.7%. *Nigella sativa* contained in Poly-Phytopharm inhibits the *cyclooxygenase* and *5-lipoxygenase* pathways from arachidonic acid metabolism, causes a decrease in thromboxane and leukotriene synthesis.14 Leukotriene

receptor antagonists have been used for the treatment of bronchial asthma, besides producing a minimal bronchodilator effect and reducing bronchoconstriction due to allergens, sulfur dioxide and exercise, also has anti-inflammatory effects.[10] *Nigella sativa* can also prevent the release of histamine from mast cells and basophils and has an anti-inflammatory effect, which then has a therapeutic effect on asthma.[15] Koshak *et al.* previously studied the effects of *Nigella sativa* extract administration to asthma patients for four weeks, at the end of the study it was shown that there was a significant increase in the average ACT score in the treatment group compared to the placebo group.[6]

Poly-Phytopharm Decreases Blood Eosinophil Levels

The administration of Poly-Phytopharm can reduce blood eosinophil levels. Similar to the results of Koshak *et al*. 's study, the administration of *Nigella sativa* extract significantly reduced blood eosinophil counts compared with placebo in stable, uncontrolled asthma patients. However, in their study, the dose of *Nigella sativa* given was higher and without a combination with other extracts.[6] Our study result was different from the effects of intervention in other previous studies with allergic rhinitis subjects, where there was no significant decrease in the percentage of blood eosinophils after *Nigella sativa* extract administration. In this study, it did not use absolute eosinophil levels, and the percentage of eosinophils before the treatment was in the range of normal values.[16]

The anti-inflammatory effect contained in Poly-Phytopharm can cause the decrease in eosinophils. *Nigella sativa, Kleinhovia hospita, and Curcuma xanthorrhiza* have anti-inflammatory effects through different pathways. Anti-viral, antibacterial, and immunity improvement effects (immunoglobulins in *Ophicephalus striatus)* can reduce the likelihood of new inflammation and exacerbations with triggers of infection.[17] *Thymoquinone* was stated to suppress the expression of IL-5.7 IL-5 will limit eosinophil recruitment and some inflammation mechanisms in the airways of asthmatic patients.

Poly-Phytopharm Increases Serum miR-126 Relative Expressions

Our study showed a significant increase in the relative expression of serum miR-126 in line with clinical improvement and decreased blood eosinophil levels after supplementation with *Nigella sativa, Kleinhovia hospita, Curcuma xanthorrhiza*, and *Opiocephalus striatus* extract for 12 weeks especially in subjects with initial blood eosinophils $\geq$ 300 pg/mL. It is possible that the increase in the relative expression of serum miR-126 results in negative feedback, resulting in a decrease in inflammation characterized by a reduction in blood eosinophil levels and an improvement in control of asthma symptoms characterized by an increase in ACT scores.

Previous studies involving 80 asthmatic children and 80 healthy children, assessed the relative expression of miR-126 in peripheral blood. They concluded that the relative expression of miR-126 increased in the peripheral blood of children with acute asthma, nevertheless miR-126 could be used as a potential serological marker in the diagnosis and assessment of asthma.18 However, in these studies, it did not assess the effect of a treatment or intervention to changes in the relative expression of miR-126 as was done in our research.

Another study in asthma patients involving 150 adults as subjects consisting of non-asthma, non-persistent asthma, and persistent asthma subjects, concluded that ten miRNAs regulate inflammation where downregulation was found in 5 miRNAs, miR-18a, miR-126, let-7e, miR-155, and miR-224. Upregulation was found in 5 other miRNAs, miR-498, miR-187, miR-874, miR-143, and miR-886-3p. In the study, besides observing miRNA expressions, researchers also measured airway mucosal eosinophil levels.[19]

Our study has several limitations. The number of subjects is relatively small because of the limited time of the research. The researchers did not carry out a specific analysis on the onset of asthma symptoms presentation and the duration of controller use. The last, only use one companion biomarker for miR-126, which is blood eosinophil levels.

CONCLUSION

This study proves that the administration of Poly-Phytopharm containing a combination of *Nigella sativa, Kleinhovia hospita, Curcuma xanthorrhiza, and Ophiocephalus striatus* extract can improve asthma control with increase ACT score, reduce blood eosinophil levels and increase the relative expression of serum miR-126 on not fully controlled stable asthma patients.

Acknowledgements

The author would like to thank PT. Royal Medicalink Pharmalab and the Department of Education and Research of Dr. Saiful Anwar Hospital Malang who helped research fund.

Conflict of Interest

The author states that there is no conflict of interest. PT. Royal Medica Pharmalab produces medications for research, and the company did not provide input on either the research design or the interpretation of the research results.

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ACCORDANCE LEVEL OF TUBERCULIN SKIN TEST (TST) EXAMINATION AND T-SPOT.TB, AND SENSITIVITY AND SPECIFICITY OF TST AND T-SPOT.TB IN DETECTING LATENT TB INFECTION IN HEALTH CARE WORKERS (HCWs).

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ABSTRACT

Background: One of the 10 WHO End TB Strategy priority indicators is the target number above 90% for the coverage of ITBL treatment. Health Care Workers (HCWs) have a greater risk of TB than the general population. There is no gold standard for diagnosis of ITBL, TST and IGRA are used to diagnose ITBL.

Objective: This study was to determine the accordance level, sensitivity, and specificity of TST and T-SPOT. TB examination. This study determine the prevalence of ITBL and the correlation between the location of work and the length of working as a HCW.

Methods: Cross sectional study design in Dr Moewardi Hospital Surakarta in December 2018. A blood sample of each HCWs was taken for T-SPOT.TB and subsequently performed the TST.

Results: Research subjects were 30 respondents; 15 health workers in the pulmonary ward and 15 administrative officers in the ward. The accordance level of TST and T-SPOT.TB is substantial ($k=0.603$, $p<0.001$). Sensitivity and specificity of T-SPOT.TB with close contact as gold standard were 60% and 86.7%, TST with close contact as gold standard were 33.3% and 93.3%. There was significant correlation between location of work in lung care ward with T-SPOT.TB ($r=0.436$ and $p=0.008$ ($p<0.05$)).

Conclusion: The accordance level between TST and T-SPOT.TB was substantial. Both have a specificity above 85%, T-SPOT.TB has a slightly better sensitivity than TST, this might be related to the booster phenomenon of TST examination in HCWs and in this study it was not followed.

Keywords: TST, T-SPOT.TB, LTBI, HCWs, Booster phenomenon.

CORRELATION BETWEEN CHRONIC OBSTRUCTIVE PULMONARY DISEASE TOWARDS THE INCIDENCE OF GASTROESOPHAGEAL REFLUX DISEASE

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ABSTRACT

Chronic Obstructive Pulmonary Disease (COPD) is a lung disease characterized by an air flow barrier that is not fully reversible and progressive. Many of the extra-lung effects on COPD are considered because it is interlaced by systemic inflammation. Systemic effect of COPD as a sign there are other comorbide conditions. Gastroesophageal Reflux Disease (GERD) is one of the most commonly found gastrointestinal conditions in general population and is known as one of the comorbidities of COPD. This research aimed to find out if there was a correlation between COPD to the incident of GERD. Observational analytic with a cross sectional approach was performed in this research. The sampling techniques used were simple random sampling. The research data was collected from the medical record data in Puri Medika Hospital. The results of the data processing on 50 samples with COPD diagnosis at the age of 40-70 years and severity from moderate to severe showed 68% of GERD incidence rate. The bivariate test results using chi-square ($p = 0,002$) conclude a significant correlation between COPD towards the incidence of GERD.

Keywords: *Chronic obstructive pulmonary disease* (COPD), *Gastroesophageal Reflux Disease* (GERD)

LEUKOCYTOSIS AS HEMATOLOGIC PARANEOPLASTIC SYNDROME IN NEW CASES OF LUNG CANCER AT THE NATIONAL RESPIRATORY REFERRAL HOSPITAL : FIRST INDONESIA DATA

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ABSTRACT

Background: Lung cancer could have signs and symptoms caused by paraneoplastic syndromes. Those paraneoplastic syndromes could involve hematologic system, one of them is leukocytosis. Research regarding hematologic paraneoplastic syndromes in lung cancer in Indonesia to date is unavailable.

Methods: This analytical cross-sectional study involved new cases of lung cancer admitted to the thoracic oncology clinic in National Respiratory Referral Hospital Persahabatan Jakarta Indonesia between September 2018 and February 2019. Samples were those who met the inclusion and exclusion criteria taken by means of total sampling.

R**esults:** About 136 samples were included in this study and had a mean age of 56.7+11.4 yo. Most of them were male (78.7%), had normal nutritional status (42.6%), had smoking history (75.0%), had moderate Brinkman Index (52.0%), were squamous cell carcinoma (SCC) (39.7%), had presented as advanced stage (83.8%), and had performance status <2 (94.1%). Paraneoplastic leukocytosis (39.0%) was associated with male sex and smoking history. Paraneoplastic neutrophilia (51.5%) was related to male sex, smoking history, and SCC histological type. Paraneoplastic hypereosinophilia was only 2.9% and not related to any characteristics.

Conclusion: Leukocytosis was a quite common hematological finding as paraneoplastic syndromes found in lung cancer.

Keywords: *paraneoplastic syndrome in lung cancer; paraneoplastic leukocytosis, paraneoplastic neutrophilia, araneoplastic hypereosinophilia*

IMMOBILIZATION AS AN APPARENT ETIOLOGY OF ICU-RELATED DELIRIUM IN PERSAHABATAN HOSPITAL, JAKARTA

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ABSTRACT

Introduction

Prior studies had shown how early detection of ICU-related delirium leads to better prognosis. Amongst many assessments tools, Confusion Assessment Method in Intensive Care Unit (CAM-ICU) is well validated and reliable. Chronic immobilization contributes to delirium and deconditioning of organ systems, leading to organ failures which constitutes to worsening delirium. This study is aimed to reveal the current proportions of delirium etiologies in the ICU, adhering to the early mobilization campaign from the Medical Rehabilitation Department.

Methods

Cross sectional observation was done on all ICU patients with the presence of delirium (positive CAM-ICU) in March 2019 regardless of mechanical ventilation utilization. Sedated patients and RASS score below -3 are excluded. Data was primarily and secondarily obtained regarding delirium etiologies. THINK Delirium mnemonic (Toxic conditions and new organ failure, Hypovolemia/Hypoxia, Immobilization/Infection/ Inflammation, Nonpharmacologic factors, K+ and other electrolyte disturbances) were used to categorize etiologies. Secondly, the presence of multi organ deconditioning syndromes were also recorded to further stratify immobilization etiologies.

Results

The study recruited 7 ICU cases with RASS ranging from 0 to -2, majority of medical diagnosis, and 2 surgery cases. All 7 cases had both nonpharmacologic factors (no noise control, inadequate sleep hygiene), and immobilization. Regarding deconditioning amongst the observable cases, gastrointestinal deconditioning predominates in all cases, followed by joints, and cardiovascular.

Discussion

Previous studies showed evidence that sleep disorder in ICU would cause longer sleep during daytime, without altering total sleep time. This affects exercise sessions during daytime, and thus interferes rehabilitation goals. Its correlation to immobility is still intriguing. It could also be seen that the presence of delirium would only allow passive interventions, hence different goals should be administered aside from weaning and mobilization successes.

Conclusion

These results reflected the importance of delirium screening in ICU, hence allowing physiatrist to adjust achievable rehabilitation goals.

ABTRACT POSTER

THE NORMAL VALUES OF MAXIMAL INSPIRATORY PRESSURE AND MAXIMAL EXPIRATORY PRESSURE IN INDONESIAN HEALTHY YOUNG ADULT: A PRELIMINARY STUDY

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ABSTRACT

Background
The strength of the respiratory muscles is showed by Maximal Inspiratory Pressure (MIP) and Maximal Expiratory Pressure (MEP) measurement. Until today, Indonesia has no normal values of MIP and MEP.

Material and Methods
A preliminary cross-sectional study with consecutive sampling that included 17 healthy young adult (normal IMT and spirometry, no scoliosis nor history of smoking, age 18-40 years old). The MIP and MEP values were measured using manometer for respiratory muscles (microRPM®). The data was obtained from February-March 2019 and was analyzed by using SPSS v.20. Results will be presented as mean and SD.

Results
The average MIP value among the subjects was 79.83±29.96 cmH2O in male and 59.81±16.01 cmH2O in female. The average MEP value was 72.67±22.74 cmH2O in male and 67.82±29.16 cmH2O in female. Both averages were higher in male than female.

Table 1. Mean Values of MIP and MEP in Male and Female Subject

Gender	MIP (cmH_2O)	MEP (cmH_2O)
Male	79.83±29.96	72.67±22.74
Female	59.81±16.01	67.82±29.16

Conclusion
Maximal respiratory pressure values were higher than those found in Singapore, except maximal expiratory pressure in male. However, maximal respiratory pressure values for both gender are lower than in Colombia. Further study with larger sample are needed for better explanation of this result.

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CLINICAL PROFILE OF SUPERIOR VENA CAVA SYNDROME WITH LUNG CANCER IN PERSAHABATAN HOSPITAL

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ABSTRACT

Background

Oncological emergencies are a group of condition that occurs as a direct or indirect result of malignancy or its treatment that are potentially life-threatening such as superior venous cava obstruction. Diagnostic procedure often postponed until resolution of respiratory complication. Emergencies treatments may increased morbidity and mortality.

Methods

This researched collected secondary data in 2017 from radiotherapy unit and oncology clinic. We recruited 56 patients lung cancer with superior venous cava syndrome (SVCS) in Persahabatan Hospital, Jakarta-Indonesia. Our primary objective was to gain clinical profile

Result

Thirty six patient lung cancer with clinically common physical of SVCS and has been confirm withradiological findings are male dominant with 91,6% non small cell lung carcinoma (NSCLC). Predominanty are former smoker 80,5% with medium (33,3%) and high (27,7%) Indeks Brikmann. There were 61,1% patient died with one year survival.

Conclusion

Thoracic oncologies present with SVCS is most frequently with underlying cause of malignancy. Multidiciplinary care will maximize treatment because of the poor overall prognosis and palliatif focus of treatment.

Keywords : emergency oncology, superior vena cava syndrome, lung cancer, radiotherapy

ANYER "TSUNAMI LUNG": CASE RERPOT

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ABSTRACT

Background
Respiratory disease deemed biggest menace in tsunami disaster's aftermath. On December 22, 2018, tsunami struck off the coast of Anyer, Banten.

Case Report
A 33-year-old woman, tsunami Anyer survivor came to emergency department with non-productive cough and shortness of breath since one day before admission. She had history of multiple sea water aspiration during the disaster. There was no chest trauma. On physical examination, She was tachypnea with respiratory rate 28 time per minutes and 85% oxygen saturation on room air. Lung auscultation revealed rhonchi in both lungs confirmed by bilateral consolidation on chest x-ray. Blood test showed leukocytosis with white blood cell 16.530 μL. Patient was diagnosed with pneumonia aspiration. She were treated with cefoperazone-sulbactam 2 gram bid for 4 days, moxifloxacin 400 mg od, methylprednisolone 62,5mg tid, acetylcysteine 200mg tid, and nebulizer (salbutamol and bisolvon) tid. Sputum culture showed no significant bacterial growth. After treatment, follow up chest x-ray was remarkable. Blood test showed leukocytosis improvement (12.770 μL). Patient was discharged to out-patient department care.

Discussion
"Tsunami lung" patients are characterized by a residual abnormal shadow.[1] Tsunami lung occurs when people being swept by tsunami waves inhale salt-water contaminated with mud and bacteria.[2] Disaster respiratory care covers patient conditions ranging from prolonged pneumonic shadows, which are resistant to anti-bacterial antibiotics, to more recently developed allergic lung inflammation, such as hypersensitivity pneumonitis or organizing pneumonia. [1] A combination of microbes likely contributes to tsunami lung but our patient showed no significant growth of sputum. [3] Pneumonitis in combination with infection were considered. There were no guidelines therapy for pneumonia aspiration due to disaster. [4]

Conclusion
Natural disasters can result in excess morbidity and mortality due to multiple types of pneumonia.

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SUPERIOR SULCUS TUMOR : RIGHT UPPER APEX INFILTRATE IS NOT ALWAYS TUBERCULOSIS IN TUBERCULOSIS ENDEMIC COUNTRY, INDONESIA

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ABSTRACT

Background
Superior sulcus tumor makes up a clinically unique and challenging lung tumor subtype.1

Case Report
A 45-year-old male with presented to emergency department with 5 months of productive cough, fatigue, progressive weight loss, and dyspnea. Chest X-ray from 3 months prior showed right apex infiltrate. He was treated with anti-tuberculosis treatment for a week and discontinued due to drug-induced hepatitis. Two months after, chest X-ray revealed worsening infiltrate which was then treated with multiple antibiotics but no improvement. Patient experienced hemoptysis, right eye ptosis, as well as right upper arm pain and paresthesia for the last 2 weeks. Vital sign was remarkable and decreased lung sound in right apex. Chest X-ray showed larger infiltrate with deviated trachea to contralateral side. Superior sulcus tumor was diagnosed and the patient was referred to type B hospital for further investigation.

Discussion
Large number of lung cancer patients was initially mistreated for tuberculosis due to high tuberculosis prevalence and radiological similarities.2 Patient had classic tuberculosis symptoms and X-ray findings which led to the diagnosis. Superior sulcus tumor represent 3% to 5% of all lung cancers.3 It is located in the apex of the lung and invade through the apical chest wall and structures of the thoracic inlet, producing Pancoast syndrome.4 In our case, the first and second chest X-ray are indifferent from tuberculosis, however, third X-ray showed deviated trachea which was suggestive for tumor.

Conclusion
Right apex infiltrate is not always tuberculosis in tuberculosis endemic country. Careful and continuous examination are crucial for differentiation of superior sulcus tumor and tuberculosis.

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PULMONARY REHABILITATION ON CHRONIC OBSTRUCTIVE PULMONARY DISEASE WITH HISTORY OF PULMONARY TUBERCULOSIS: CASE REPORT

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ABSTRACT

Background
Pulmonary Tuberculosis (TB) in Chronic Obstructive Pulmonary Disease (COPD) increases the frequency of exacerbations and contribute to severity of patient's disability.

Case:
A 59-years-old male had recurrent cough and difficulty to remove phlegm for 1 month. He was mild smoker with history of pulmonary TB. He felt shortness of breath on climbing stairs but ADL independently. Physical examination found hyperkyphotic-thoracal, minimal phlegm and chest expansion limitation. Spirometry showed mild restrictive and moderate obstructive. After 10 months COPD rehabilitation program, he had improvement on exercise capacity (6MWD), CAT and mMRC score.

Discussion:
The pathologies process of pulmonary TB together with tobacco smoking caused lung destruction and airways limitation that develop into COPD GOLD grade 2 group C. Pulmonary rehabilitation (infrared therapy, breathing and aerobic exercise), aggregating factors avoidance and healthy diet were given. After 10 months, CAT score reduced (12 to 9) and showed improvement on cardio-respiration endurance (4.4 to 6 METs on 6MWD). However, he still had inefficiency airways clearance thus, the relaxation and breathing techniques with proper postural given to release the muscle spasm problems, control breathing during activities and improve effective cough.

Conclusion:
This case highlights the importance of pulmonary rehabilitation in COPD management with history of pulmonary TB. Nowadays, the program is still ongoing process to improve the airways clearance and prevent exacerbation.

TUBERCULOSIS CONTACT HISTORY BETWEEN SECONDARY RIFAMPICIN RESISTANT TUBERCULOSIS AND PRIMARY RIFAMPICIN RESISTANT TUBERCULOSIS PATIENTS

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ABSTRACT

Background: Dr.Moewardi Hospital has been managed patient with primary rifampicin resistant (RR) and secondary rifampicin resistant (RR) TB since 2017. This study aimed to determined differences of contact history with TB patients, HIV status, DM comorbidities and presence or absence of BCG scar between primary RR TB patients and secondary RR TB.

Material and Method : A retrospective study on medical record data of Tuberculosis RR patients at Dr.moewardi Hospital Surakarta from January 2017 until March 2019 was conducted. Study subjects 160 RR TB patients performed. Patient with primary Tuberculosis category 10 and secondary tuberculosis category 1 to 9 were include in our study. Univariate and Bivariate analyses were applied to asses the normal distribution data and the differences in the study variable respectively.Multivariate analysis using logistic regression on variables was used if P <0.250

Results : Bivariate analysis on secondary and primary RR Tb reveded that history of Tb contact, HIV, Diabetes Mellitus, and BCG Scar were p = 0.002 (p <0.05) ; OR = 3.794 (1,563-9,208), p = 0.051 (p> 0.05) ; OR = 0.139 (0.014-1.375), p = 0.863 (p> 0.05) ; OR = 1.076 (0.470-2.464), p = 0.440 (p> 0.05) ; OR = 0.722 (0.316-1.651). Multivariate analysis showed that history of Tb contact was dominated factor in secondary RR Tb patient (p = 0.003 ; OR = 3.860).

Conclusion : there is significant difference in contact Tb history between secondary and primary RR Tb. The history of contact Tb is 3.8 folds more likely to be encountered in secondary RR Tb.

Keywords : TB contact History,Primary RR TB, Secondary RR TB

SIX MINUTES WALKING DISTANCE IN A PATIENT WITH COPD GROUP C WITH GOOD ADHERENCE: A CASE REPORT

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ABSTRACT

Background

Six minutes walking test (6MWT) is a standardized submaximal exercise testing that entails measurement of distance walked over a span of six minutes. The 6MWT is an objective measurement to assess functional capacity of a person. It developed in an attempt to accommodate patient with respiratory disease for whom walking 12 minutes was too exhausting. In some study known that there is an improvement to 70 meters in the 6MWT after intervention in patient with COPD. Adherence to therapeutic interventions, including a healthy lifestyle and regular exercise programme, are crucial in the management of COPD. Some studies shows the benefits of pulmonary rehabilitation regress towards baseline values after 6–12 months.

Methods

We observed one patient with COPD Group C according to GOLD classification, and evaluated the increase of walking distance in 6MWT after included into Pulmonary Rehabilitation in Persahabatan Hospital, consist of effective breathing exercise, chest therapy, series of treadmill endurance exercise, two times a week and added also functional walking exercise as a home program.

Result

There is an improvement of his 6 MWT reached after 6 months routine endurance exercise with good adherence. At the baseline data, he had a 366 meters of walking distance (62,3 % prediction) which increase significantly to 490 m distance in 6MWT (83% prediction) at February 2019, and meets the minimal clinical important difference threshold.

Conclusion

6MWT can be used to measured functional capacity in patient with COPD. Showed improvement after routine exercise

THORACIC SEQUELAE AND OBSTRUCTIVE AIRWAY DISEASE IN TREATED PULMONARY TUBERCULOUS PATIENT : A CASE REPORT

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ABSTRACT

Up to half of post pulmonary tuberculosis patients have some continues structural damages despite microbiologic has been cured. A variety sequelae can occur in the pulmonary and extrapulmonary portions of the thorax. Structural changes lead to pulmonary impairment from moderate to severe. Pulmonary dysfunction post tuberculosis can increase the risk of obstructive airway disease and death from respiratory causes. A sixty four years old man, unemployed and non smoker, presented with three days of progressive dyspnea. He had history of successfully treated pulmonary tuberculosis seven years ago, repeated hospitalization, and corticosteroid inhaler use. Physical examination showed underweight body mass index, respiratory rate 32 times per minutes, SaO2 94%, crackles and wheezing found bilateral in upper lung field. Laboratory work up revealed leukocytosis, alkalosis respiratory without hypoxemia on arterial blood gas analysis, and bacteria was not detected on gene expert test. An active pulmonary tuberculosis with secondary infection and pleural thickness were found on chest x ray. CT thorax without contrast showed a bronchiectasis with pneumonia,panlobular emphysema,and schawrte with bilateral pleural effusion.He received a supportive and symptomatic treatment and showed clinical improvement. At follow up, spirometry test revealed a mixed pattern ventilatory defect. A tuberculosis sequelae and complications are impacting the quality of life of affected individuals. Furthermore, it would create a significant burden on healthcare in developing countries. Therefore need more prevention, assessment , and care for post tuberculosis patients.

Keywords : pulmonary tuberculosis, thoracic sequelae, obstructive airway disease

THE ADHERENCE OF PULMONARY REHABILITATION PROGRAM IN PATIENT WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE : A CASE REPORT

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ABSTRACT

Background
Pulmonary rehabilitation (PR) program was developed to optimize the management of patients with chronic obstructive pulmonary disease (COPD). Patient's adherence to PR program is essential to optimize disease management.

Methods
A 57 old man admitted to hospital due to shortness of breath and lot of sputum production and was diagnosed with COPD. After discharged, he was referred to rehabilitation clinic and assessed with low cardiorespiratory endurance, disturbance of chest mobility and sputum retention. He was given chest mobility exercise, aerobic exercise, active cycled breathing technique and chest physiotherapy. The evaluation was done every month after the exercise program to assess functional capacity including 6 minute walking test (6mwt), borg scale, CAT score, peak cough flow (PCF) and peak flow rate (PFR).

Results
In the first 3 months of the program, the patient came regularly and there was improvement in Borg scale, CAT score, PFR, PCF and 6mwt results. After that he did not come regularly for evaluation and there were variation of all the parameter results. After not came to exercise for 4 months, there were increase in rating of perceived exertion (RPE) of Borg scale from 8 to 11 and also decline in distance of 6mwt from 359 meter to 327 meter.

Conclusion
Adherence in exercise may influence the outcome of pulmonary rehabilitation program, in this case affecting Borg scale and 6mwt results.

THE ROLE OF CHEST ULTRASONOGRAPHY VERSUS X-RAY FOR DETECTION OF TRAUMATIC PNEUMOTHORAX : AN EVIDENCE-BASED CASE REPORT

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ABSTRACT

Background: In the event of natural disasters, early diagnosis of blunt chest trauma is imperative in decreasing mortality rate. One of the most common blunt chest injury is pneumothorax. Chest X-ray (CXR) has been routinely used in patients with chest trauma but its accuracy in detecting pneumothorax is satisfactory. Chest ultrasonography (CUS) can be used as an alternative in resource limited setting, albeit operator dependent.

Background: To compare the diagnostic accuracy of CUS and CXR in detection of traumatic pneumothorax.

Methods: A comprehensive search was conducted on PubMed, Scopus and Cochrane. The selection of title and abstracts was conducted using strict inclusion and exclusion criteria. The selected articles were evaluated using standardised critical appraisal tool.

Results: 2 meta-analyses with comparable validity were included in the final review. In the more recent study, pooled sensitivity and specificity were 85% and 99% respectively for CUS, whereas 46% and 99% respectively for CXR. In the other study, CUS was 90% sensitive and 99% specific while CXR was 50% sensitive and 99% specific.

Conclusion: Present meta-analyses demonstrate consistently that diagnostic accuracy of CUS is superior to CXR for detection of traumatic pneumothorax. Ultrasound is a convenient and readily available bedside procedure, appropriate to use in disaster setting and accurate in diagnosing traumatic pneumothorax.

Keywords: *Chest Ultrasonography, Chest X-ray, Pneumothorax, Diagnostic Test*

PLATELET COUNT AND NEUTROPHYL LYMPHOCYTE RATIO AS A PREDICTOR SEPSIS IN PATIENT WITH COMMUNITY ACQUIRED PNEUMONIA

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ABSTRACT

Background : community acquired pneumonia (CAP) represents a significant infection burden worldwide, and its complicated by sepsis. According the recent studies, biomarker such as neutrophyl to lymphocyte ratio (NLR) and platelets count have been proposed as indicators of systemic inflammation and infection. In our study, we aimed to evaluate NLR and platelet count used to predictor sepsis in patient with CAP and prognosis mortality patients with sepsis.

Material and method : We performed a retrospective study of 86 patients hospitalized with CAP at the Dr. Moewardi hospital in Surakarta, between June 2018 and December 2018. Predictor variables were platelet count and NLR. Neutrophyl to lymphocyte ratio was defined as absolute neutrophil count divided by lymphocyte count. Univariate and Bivariate analyses were applied to asses the normal distribution data. Bivariate analysis using logistic regression on variables was used if $P < 0.05$.

Results : Bivariate analysis on platelet count and NLR in patient with CAP were $p = 0.464$ ($p > 0.05$), $p= 0.320$ ($p > 0.05$). A significant association between CAP with sepsis and prognosis mortality was found ($p= 0.004$).

Conclusion: Platelet count and NLR can not be used as an predictor sepsis in CAP. Community acquired pneumonia with sepsis have a high risk for death.

Keywords: platelet count, neutrophyl lymphocyte ratio, sepsis, community acquired pneumonia

MAIN CAUSE OF DEATH IN TRAUMATIC TENSION PNEUMOTHORAX

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ABSTRACT

Introduction: Traumatic tension pneumothorax is the accumulation of air under pressure in pleural space caused by either blunt or penetrating thoracic trauma. Traumatic tension pneumothorax accounted about 5.4% of major trauma patient. Tension pneumothorax develops when injured lung tissue forms a one-way valve, allowing air to enter the pleural space and prevent the air to escape naturally. Traumatic tension pneumothorax is an emergency case that leading to death. Cause of death is various and still debated.

Objective: Discuss various causes of death in traumatic tension pneumothorax.

Discussion: Several studies have investigated causes of death in trauma patients and classified them to immediate-early and late death. Respiratory failure is the most common immediate-early cause of death that counted only in 24 hours after trauma. However, circulatory failure is the most common late cause of death due to secondary results from respiratory failure. The respiratory failure mechanically caused by ipsilateral lung collapse resulting acute hypoxia. High pressure in contralateral lung disrupt cavoartrial junction and decrease the diastolic filling. These mechanisms induce low cardiac output and resulting circulatory failure. Worsening hypoxia in untreated patients can cause general vasoconstriction, which means distributive shock mechanism. The pathophysiology showed two general mechanisms of death related to cardiovascular collapse, direct by mechanical compression and indirect by worsening hypoxia. Some cases the patient came to emergency with complicated traumatic tension pneumothorax, when both respiration and circulation collapsed together. Therefore, the patient must be received treatment as soon as possible.

Conclusion: Traumatic tension pneumothorax has various mechanisms cause of death. Respiratory failure is the fastest mechanism related to death due to acute hypoxia.

Keywords: Tension, Pneumothorax, Trauma.

INHALED MAGNESIUM SULPHATE AS ADD-ON THERAPY FOR LIFE THREATENING ASTHMA ATTACK: A CASE REPORT

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ABSTRACT

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Introduction: Asthma attack is one of the common presentations to emergency room, with manifestation vary from mild to life threatening attack. While the role of SABA, SAMA, and corticosteroid has established as treatment for asthma attack, the use of inhaled magnesium sulphate is still controversial. Global Initiative for Asthma (GINA) guideline has recommend intravenous magnesium sulphate as treatment for severe asthma attack, but not inhaled magnesium sulphate.

Case Presentation: We present an interesting case of a 41-year-old man with symptoms suggestive of a life-threatening asthma attack with type 2 respiratory failure. He was treated with inhaled SABA+SAMA, inhaled corticosteroid, and systemic corticosteroid for 2 days with no improvement of clinical condition. After addition of inhaled magnesium sulphate as add-on therapy, the patient's clinical (dyspnea, respiratory rate, wheezing, use of accessory muscles) and laboratory (PaO2, PaCO2 and pH) were improved significantly within 1 day of therapy. Therapy was continued for 3 days and patients showed consistent clinical improvement.

Conclusion: Inhaled magnesium sulphate has demonstrated possible role in treating patients with life threatening asthma attack.

Keywords: Inhaled magnesium sulphate, asthma attack, add-on therapy

EARLY KEY SUCCESS OF NON-INVASIVE VENTILATION IN SEVERE ARDS: A CASE REPORT

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ABSTRACT

A 59 years old patient admitted to intensive care unit with urinary tract infection, acute kidney injuryand atrial fibrillation with rapid ventricular response. At the second day of admission in the intensive care unit patient suddenly felt dyspnea and respiratory distress that doesn't improve with nonrebreathable mask, based on the Berlin criteria patient was diagnosed with severe ARDS (PaO2/FiO2 ratio 86.9). We initiate non-invasive ventilation (NIV) in this patient. Evaluation was done by using clinical (respiratory rate, dyspnea and use of accessory muscle, consciousness), physiological (oxygen saturation, arterial blood gas analysis), ventilatory (expiratory tidal volume, minute ventilation), and cardiac parameter (blood pressure) two hours post NIV initiation. Two hours and one day evaluation post NIV in this patient shown not worsening and improvement in all parameter. Two hours evaluation is the crucial point to determine that the patient will respond well or fail. What is important in this case report is the parameter used for two hours evaluation post NIV use and the importance of these evaluation for determining the success of NIV use in ARDS.

Keywords: non-invasive ventilation, acute respiratory distress syndrome, two hours evaluation

HOME-BASED AEROBIC AND MUSCLE ENDURANCE EXERCISE IN PATIENT WITH SYSTEMIC SCLEROSIS RELATED INTERSTITIAL LUNG DISEASE

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ABSTRACT

Objective: To improve fitness in systemic sclerosis-related interstitial lung disease patient with low muscle endurance and cardiorespiration fitness.

Methods:
46-year-old female with a diagnosis of Systemic Sclerosis related Interstitial Lung Disease (SSc-ILD) had main complaint of fatigue and dyspnea while doing everyday activities. She had a serial of examinations with results as restrictive lung function with low cardiorespiratory endurance. A rehabilitation programme that consists of muscle endurance using functional index combined with a cardiorespiratory exercise in 80% intensity with repeated pulmonary functional measures during a twelve-week home programme was implied to the patient. Whilst undergoing this programme she also takes her medication regularly.

Results: Six-minutes-walk distance and forced vital capacity increased as much as 95 meters and 13,6% which indicates the improvement of cardiorespiratory fitness and restrictive pulmonary function. The improvement of muscle endurance is shown from the increased repetition of right and left shoulder flexion also right and left hip flexion as much as 16, 18, 22, 20 times respectively.

Conclusions: Components of a twelve-week rehabilitation program together, was successful with positive effects on muscle endurance, cardiorespiratory fitness and pulmonary functions.

Keywords: Systemic sclerosis, Interstitial lung disease, Rehabilitation, Muscle endurance, Cardiorespiratory fitness

SLEEP-DISORDERED BREATHING IN HEART FAILURE REDUCED EJECTION FRACTION

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ABSTRACT

Background: Heart Failure (HF) still carries high burden for patient.1 It is the end stage of many heart disease and is highly prevalent.[2] Recognizing and treating comorbidities which contributes to progression of heart failure becomes important in order to achieve good outcome. Among multiple comorbidities, an important condition that is often unrecognized is sleep-disordered breathing (SDB).3 SDB occurs in over 50% of patients with heart failure.[4] SDB may exacerbate HF condition by causing activation of neurohumoral and hemodynamic responses that are detrimental to the failing heart.5 Hence, the presence of SDB in heart failure patients may worsen the progression of HF and confers poorer prognosis for the patients.[6]

Case Illustration: A 70-years-old woman presented to emergency department with shortness of breath, orthopnea and paroxysmal nocturnal dyspnea. She is a doctor, however she has poor adherence toward her medication. She has been our regular patient in National Cardiac Center of Harapan Kita and previously catheterized with a result of normal flow coronary artery. Her echocardiography result on 2016 showed ejection fraction of 56%. Upon physical examination, patient was compos mentis, there were signs of congestion as indicated by increased in respiration rate of 27 times per minute, fine rales in both side of 1/3 basal of the lungs and bilateral ankle edema. The electrocardiography examination demonstrated Atrial Fibrillation with QRS rate 120 beats per minutes. The chest radiography showed cardiomegaly with 75% cardiac thoracic ratio with increased of pulmonary vascular markings. Her current echocardiography examination showed all chambers dilatation, poor echo window, reduced LV systolic function with LVEF 22% and global hypokinetic.

It was apparent that the patient had signs and symptoms of Acute Decompensated Heart Failure with reduced ejection fraction (EF 22%). She was hospitalized and given optimum heart failure therapy such as Furosemide, Candesartan, Bisoprolol and Spironolactone. However, after optimal dose of diuretics, her symptoms did not subside even though she had appeared euvolemic. On the fifth day of hospitalization, during ward visitation, the patient was found sleeping with no signs of breathing and she was also snoring. She then suddenly woke up after 20 second and quickly return back to sleep. Through further observation and evaluation, it was clear that the patient was experiencing sleep apnea. She was then discharged to undergo polysomnography. Based on the polysomnography result, the apnea and hypopnea event lasted about 12.5-40.5 seconds during sleep at night with AHI (Apnoehypopnoe index) 29. She was immediately start on continuous positive airway pressure (CPAP). Upon discharge she was given optimal HF medication such as Uperio, Spironolactone, Bisoprolol and Furosemide. Patient was recommended to use CPAP routinely every night. After 2 weeks of routine used of CPAP, her condition is improved. Patient also admitted she has never slept better. She is now able to continue her profession as a GP

	Parameters	Time / % (Index is by divided by TST)			
Sleep Report	Time in Bed (TIB)	267.0	Min		
	Sleep Period of Time(SPT)	171.5	Min		
	Total Sleep Time(TST)	171.5	Min		
	REM	0.0	Min	0.0	%
	Sleep N1	121.0	Min	70.6	%
	Sleep N2	49.0	Min	28.6	%
	Sleep N3	1.5	Min	0.9	%
	Light off – Sleep onset	85.5	Min		
	REM Latency (From Sleep onset)		Min		
	Sleep Efficiency (TST / TIB)×100	64.2	%		
	Arousal Index	15.4	Times/hour		
Respiratory Report	Total Number of Apnea and Hypopnea	83	AHI	29.0	/h
	Number of Apnea	1	AI	0.3	/h
	Type of Apnea Central (CA)	0		0.0	/h
	Obstructive (OA)	1		0.3	/h
	Mixed (MA)	0		0.0	/h
	Longest duration of Apnea	12.5	Sec.		
	Number of Hypopnea	82	HI	28.7	/h
	Longest duration of Hypopnea	40.5	Sec.		
Other	SpO2 Mean	92	%		
	SpO2 Lowest	88	%		

Figure 1 Polysomnography result

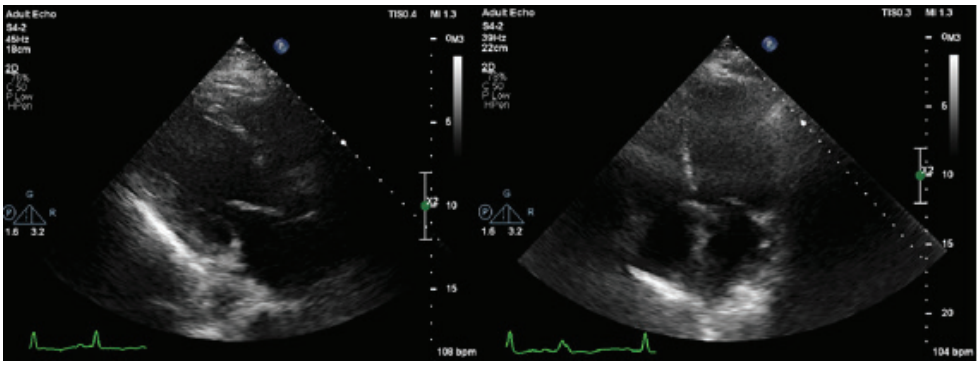

Figure 2 Echocardiography

Conclusion: We reported a case of 70 years old obese female doctor who came to the emergency room with shortness of breath on exercise, PND, orthopnoea and ankle oedema. Initially, she was diagnosed with acute decompensated heart failure with reduced ejection fraction with normal coronary and atrial fibrillation. Furthermore, following further evaluation, we found a comorbid sleep-disordered breathing that cause deterioration of the left ventricular function. Under optimal HF medication and treatment using CPAP, her condition is improved. Currently, patient has better sleep quality, therefore able to perform her daily profession as a GP. Unfortunately, poor adherence toward guidelines directed medical therapy as well as CPAP continues to be a major problem in this patient.

Keywords: heart failure, sleep-disordered breathing, sleep-disordered breathing in heart failure, obstructive sleep apnea, CPAP in heart failure

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The 22nd International Meeting on Respiratory Care Indonesia
Jakarta on August 13rd - 15th, 2020

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